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Thirty-ninth Annual Meeting

THE AMERICAN ACADEMY OF PERIODONTOLOGY

Palace Hotel, October 1955

San Francisco, California

Spring Seminar

May 2, 3, 4, 1955

Ann Arbor, Michigan

B. O. A. THOMAS, B.A., D.D.S., M.S., PH.D.

Chairman Scientific Sessions, Thirty-ninth Annual Meeting

American Academy of Periodontology

Dr. Thomas has, since June 1953, been on military leave from his position as Professor and Executive Officer of the Department of Periodontology, University of Washington, Seattle. He is serving as Commander, Dental Corps, U. S. Navy, at Treasure Island, San Francisco. During 1953-54 he was on duty as Consultant-Instructor in Periodontics at the Naval Dental School, National Naval Medical Center, Bethesda, Md. Dr. Thomas' time in civil life is divided between teaching and private practice, limited to Periodontics. In addition to holding membership in numerous dental and other scientific organizations, he is a diplomate and member of the American Board of Periodontology, and a member of the Council of the American Academy of Periodontology. Cdr. Thomas expects to return to civilian status on July 1, 1955.

THE JOURNAL OF PERIODONTOLOGY

APRIL, NINETEEN HUNDRED FIFTY-FIVE

The Defense Mechanism of the Gingiva*

by SUMTER S. ARNIM, D.D.S., PH.D.,† AND R. T. HOLT, D.D.S.,‡ *Temple, Texas*

THE purpose of this paper is to report certain changes observed in the gingiva, and thoughts concerning them. The changes are found wherever bacterial masses accumulate on or adjacent the periodontal tissues. They are not seen when bacterial masses are absent. The nature of these changes reveal the injurious effect of the organisms and the reaction of the tissues to their presence. These reactions vary from edema with separation of connective tissue fibers, proliferation of a few cells of basal epithelium, hyperemia and some cellular exudation to extensive epithelial proliferation, thickening, wild downgrowth into the corium, parakeratosis, necrosis, ulceration, hemorrhage, massive cellular exudation, disintegration and obliteration of connective tissue fibers, bone resorption and degeneration of the entire supportive mechanism.

MATERIALS AND METHODS

Serial sections of partly demineralized human, rat and monkey jaws, containing large amounts of enamel remains in natural relation to the other tissues, provide the material for this study. As the bacterial masses are retained in their customary position on enamel, gingiva, and between the gingiva and the tooth, the defense mechanism initiated by their presence is specifically delineated.

REVIEW OF THE LITERATURE

A. Toxic Effect of Bacteria

Graham[1], 1937, studied the toxic effect of sterile filtrates from periodontal pockets on remote structures by injecting them into various animals. His observations suggested substances found in periodontal lesions were highly toxic and tended to injure other organs.

Recent studies by Bibby[2] and Schultz-Haudt[3,4] give evidence that bacteria

*This study was supported by a United States Public Health Service Research Grant.

Photomicrographs for this study were made by R. M. Christopher and R. D. MacIntyre, Veterans Administration Center, Temple, Texas.

The rat, monkey and autopsy specimens used in this study are from the Dental Study Unit, Yale University School of Medicine, B. G. Anderson, Director; and the Dental School of the Medical College of Virginia, Harry Lyons, dean.

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found on teeth at the gingival margin and in the sulcus may play a significant role in the causation of periodontal disease. Among the bacterial factors mentioned are hyaluronidase, which was found to be present in all cases of generalized chronic gingivitis, and a separate factor capable of causing transient hyperemia in sensitized rabbit skin. Large increases in numbers of fusiform bacteria, spirochetes and vibrios were seen when compared with smears from clinically normal patients. The increases were greatest in the gingival crevice.

B. Tissue Response

The presence of lymphocytes in the healthy gingival epithelium has been described by Cattoni.[5] He also discusses the role of various wandering elements in this tissue. Fehr and Muhlemann[6] find more inflammatory cells in the epithelial attachment than in the oral epithelium of the rat papilla.

The histologic changes occurring in the gingiva and especially those of the reticuloendothelial system have been of special interest to Aprile.[7,8] In her opinion there are innumerable reticulo-histiocytes, both fixed and movable, distributed in the gingival connective tissue. They, in addition to other phagocytic cells in the area, clear the gingival corion of all kinds of foreign bodies, necrotic cells, refuse, blood elements and probably microorganisms. In addition, the reticulo-endothelial system assists in repair of the injured tissues. All this activity is related to the defensive function and supports Carranza's and Erausquin's[9,10] concept of "paradencio de proteccion" of the crest mucosa that covers the necks of the teeth.

C. Anatomic Factors

1. Epithelial Tissues

Studies by Baume,[11] using the phase contrast microscope, describe the arrangement existing between the epithelial attachment and the tooth as viewed with this technic. He is of the opinion that ". . . stability and firmness of the dento-epithelial junction depend primarily upon the longevity of the attached epithelium rather than the vitality of the tooth surface. Prevention of epithelial detachment thus commences with the preservation of the vitality of the superficial epithelium and its attaching tono-fibrils." Careful study of Baume's contributions indicate he is of the opinion a fibril network exists between the surface cells and the tooth substance but that it is relatively easy to separate this surface epithelium from the tooth by applying some force. In this respect Baume is in agreement with Hatton[12] and Toller,[13] who state that the mechanical attachment of epithelium to enamel is very slight, and that it does not justify any great faith in the effectiveness of the epithelial attachment. Zander[14] has devised a method for studying the epithelial attachment that gives promise of clarifying the problem. It permits a correlation of clinical observations with histologic findings. A strip of cellulose acetate film is inserted into the gingival sulcus and sections are made after

Fig. 1. Bucco-linguo-axial section of erupting upper left third molar. E, enamel remains; B, Buccal gingiva. (84X).

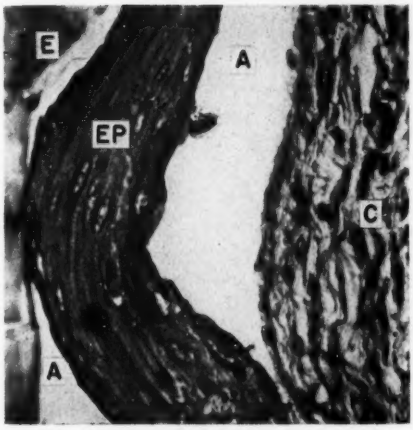

Fig. 2. High-power of buccal gingiva with artefacts, A; enamel remains, E; epithelium, EP and underlying healthy connective tissue, C. (400X).

a given period of time. The epithelium cellulose acetate junction in a dog's incisor looks like an enamel epithelial attachment.

Waerhaug[15,16] says it is of great significance to elucidate both theoretically and practically, the relation between the clinical and histologic concepts of the gingival pockets. He has conducted extensive research demonstrating the lack of firm organic union between enamel surfaces and gingival epithelium as well as the reaction of the gingiva to various irritating agents. His studies indicate the generally accepted theories concerning "epithelial attachment" with their implied clinical significance are open to serious question. Butcher's[17] experiments with replanted monkey teeth also throw light on this subject.

2. Connective Tissues

The exact arrangement of the connective tissues in the marginal gingiva and their response to injury and treatment have been described previously.[18,19] The importance of the circular fibers in the crest was emphasized. It was shown that these fibers were in greatest numbers when little cellular infiltration was seen. The presence of a dense circular band was considered an indication of gingival health. The connective tissues of the marginal gingiva as well as the periodontal membrane have been studied in great detail by Feneis[20] and Frohlich.[21] They also feel this tissue is a significant factor in maintenance of gingival integrity.

RESULTS

Figure 1 is a low power section of buccal gingiva of an erupting left third molar from a 20 year old female who died with tuberculous meningitis. Descriptions of the sections shown apply to the entire serial. The ones chosen for photomicrographs are representative of the whole block. This section contains typical marginal gingiva with few bacteria present. The epithelium covering the mouth side is thick and almost identical with that seen on the mucosa of the cheek. Little, if any, round cell infiltration is found underlying the epithelium in the connective tissue. The rete peg ridges are well developed, there is evidence of proliferation of the basal layers, migration of newly formed cells outward and partial keratinization with casting off of surface cells. The picture is one of normal mucous membrane epithelium undergoing the traditional changes of its life cycle.

As one follows this epithelium on the mouth side to the tip of the gingival margin beside the enamel surface no changes are observed. The epithelium adjacent the enamel at the crest of the gingiva has shrunken or torn from the enamel surface. No colonies of bacteria are seen at this magnification. The continuity between epithelium covering the mouth side of the gingiva and that on the enamel side is unbroken. The thickness diminishes rapidly in the cemento-enamel junction direction. At this level in the section another artefact is seen, this time the epithelium is torn from the connective tissue. The position of the tissues in relation to the enamel, as seen in the section, gives no evidence of the strength of union between cells or tissue elements.

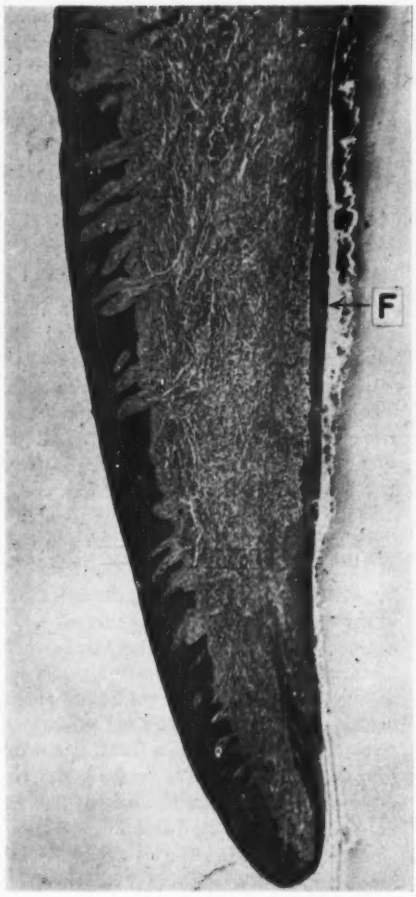

Fig. 3. Occlusal part of lingual gingiva, from same tooth seen in Fig. 1. Note bacteria and foreign material, F, between enamel and epithelium. Arrow indicates occlusal extent of Fig. 4. (84X).

Fig. 4. Lingual gingiva nearer apex than Fig. 3. (84X).

In the dense, thick connective tissue fibers of the tip of the gingiva some inflammatory exudate may be seen next to the enamel. Less is found in the gingiva on the buccal side of the tooth than on the lingual (Figs. 1 and 3). None was observed in the serial sections of the buccal gingiva from the level of Figure 2 apically. The mat of free gingival fibers is clearly defined underlying the epithelium adjacent the enamel. The body of the gingiva is composed of a dense mass of healthy connective tissue in which the circular fibers occupy a prominent part. All the fibers impart qualities of toughness, tissue tone and relative immobility to the healthy gingiva. These observations are readily confirmed by microdissection of any healthy specimen.

The lingual gingiva (Fig. 3), reaches almost to the tip of the partly erupted tooth cusp. Its crest is sharp and thin. There is evidence of an initial response of this tissue to injury. The epithelium adjacent the enamel is thickened slightly at the tip of the gingiva. A secondary cuticle extends occlusalward from the bottom of the gingival sulcus. A scattering of round cells is present in the connective tissue underlying the sulcus and adjacent the

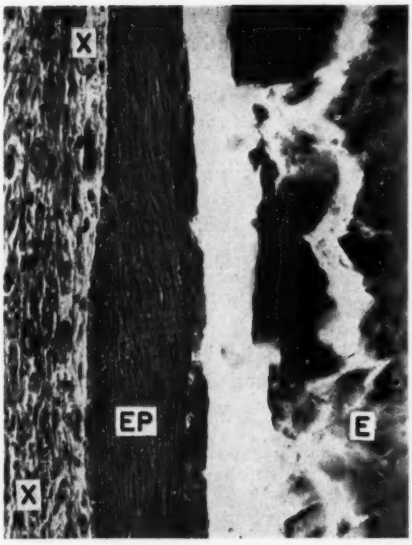

Fig. 5. High-power of foreign material, F, Fig. 4, seen between enamel, E, and epithelium, EP. Note exudate, X. (200X).

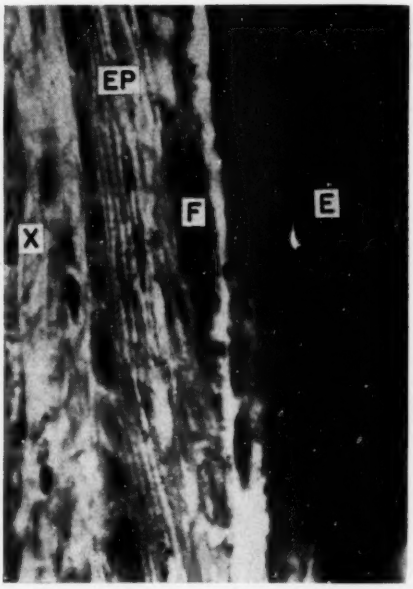

Fig. 6. High-power nearer cemento-enamel junction, circle Fig. 4, changes slight. (465X).

enamel surface. The relation between the enamel and the epithelium is disturbed in places by artefact and shrinkage. Enough enamel remains are present to give a good idea of the conditions existing prior to the preparation of the tissues for histologic study. The epithelium beside the enamel thins rapidly as the cemento-enamel junction is approached. The over all appearance is one of relatively healthy gingiva. There are few human specimens exhibiting so little change.

Dark, basophilic clumps resembling coccoid bacteria lie between the epithelial and enamel surfaces (Figs. 4, 5 and 6). They become less conspicuous as the cemento-enamel junction is approached. Higher magnification reveals leukocytic remnants within the clumps (Figs. 5 and 6). Surface cells are partly keratinized and some are cast off between the enamel and the gingiva. The germinative layer of epithelial cells underlying the bacterial clumps is prominent and evidences of developmental activity and thickening are seen (Figs. 5 and 6). There is separation and some disintegration of the connective tissue fibers in the corium. Phagocytes are prominent in the changed connective tissue and some leukocytes may be seen in the epithelium.

As the epithelium approaches the cemento-enamel junction, smaller and smaller clumps of bacteria like masses are seen. They are assumed to be bacterial due to morphologic and staining characteristics. As the clumps diminish in size the reaction in the underlying tissues diminishes in quantity.

About half way to the cemento-enamel junction one reaches an area where physiologic adaptation of the epithelium to the enamel surface is not marred by the presence of irritating elements (Fig. 7). The connective tissue fibers underlying the epithelium are dense, closely packed and contain no evidence of inflammatory response to injurious agents. The epithelial cells in the basal layer are flat and thin.

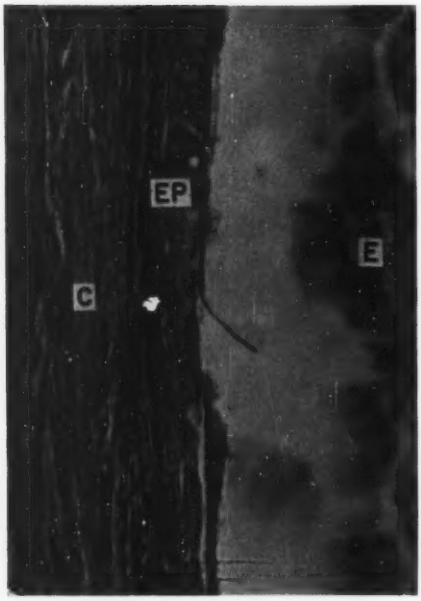

Fig. 7. High power from cervical third, oval Fig. 4, no changes, healthy gingiva. (465X).

the bacterial masses at the neck of the tooth are much larger and in some areas they are mineralized to form calculus. Adjacent the masses of calculus and bacteria the connective tissue fibers are obliterated and replaced by inflammatory exudate, fibroblasts, new capillaries and tissue fluids (Fig. 9). The edematous nature of the response to the injurious agents has led to separation of the deeper connective tissue elements. There is an intermediate region where some fibers are disintegrating and others are widely separated. The penetration of the disease into the periodontium is manifested by these changes.

The epithelium adjacent the bacterial masses is altered so much it has little resemblance to its original state. Mitotic figures are easily found in the basal layers. Long fingerlike ridges penetrate deeply into the underlying inflammatory zone. New capillaries, exudate and fibroblasts occupy the spaces between the ridges, extending almost to the surface, where they are separated from the bacteria filled gingi-

No evidence of proliferation is seen. Artefacts are observed where the surface cells of the epithelium cling to the enamel, where the enamel remains cling to the surface of the epithelium, where the primary cuticle is torn and bent toward the enamel remains, and where the enamel remains are separated from the primary cuticle (Fig. 7). None of these artefacts indicate, much less prove, that in life the epithelium is firmly attached to the enamel. Questions regarding strength of attachment are not solved by histologic sections.

In Figure 8 is seen a bucco-linguo-axial section through the mesial pit of an upper right first molar. The specimen is from a female, age 37, who died with a pulmonary embolus following childbirth. Changes characteristic of advancing periodontal disease are seen in the serial sections made from this block of tissue. The section shown is representative of the entire serial.

This case differs from the first in that

Fig. 8. Bucco-linguo-axial section, upper right first molar. CA, calculus. Note inflammation characteristic of chronic gingivitis. (12X).

val sulcus by a thin sheet of the proliferating epithelium. In many places even the few covering epithelial cells are missing and the capillaries, with surrounding exudate, are in direct contact with the bacterial masses in the sulcus.

The outpouring of tissue fluid and cellular elements into the gingival sulcus provides a rich nutrient for the bacterial masses thronging the spaces between the flabby gingiva and the tooth. The fixation of the bacteria on the tooth and within the porosities of the calculus provides a constant evergrowing source of germs, toxins and possibly other irritants which the inflammatory reaction feeds, rather than eliminates. It is this *fixation* of masses of bacteria on the tooth and in the pocket, with a constant food supply from adjoining tissues, that accounts for the characteristic persistent, chronic lesion of periodontitis. There is no natural defense mechanism with the power to remove the injurious agents once they colonize in this manner. Should bacterial masses lodge within folds of mucous membrane and colonize therein similar changes may occur.

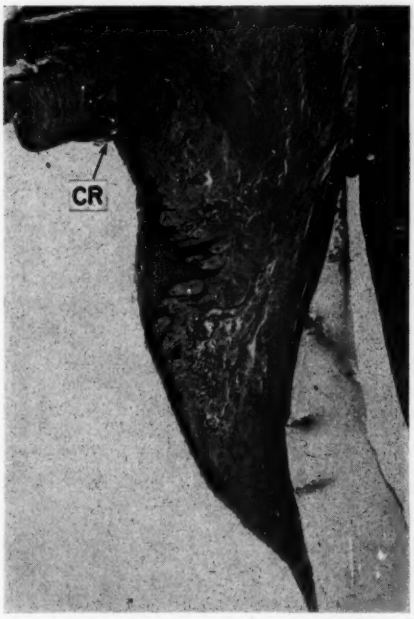

Fig. 10. Bucco-linguo-axial section of an upper right third molar from a 22 year old male. CR. crypt. (56X).

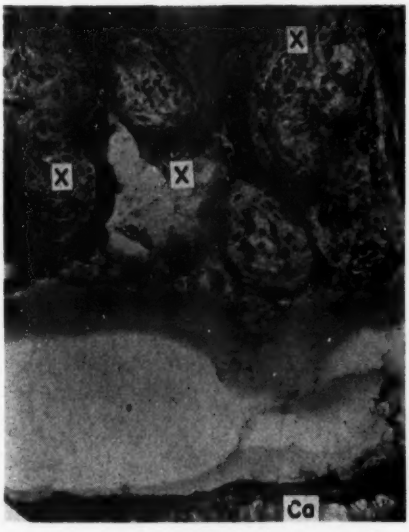

Fig. 9. Increased magnification of rectangle in Fig. 8 showing ulceration, exudation and proliferation adjacent bacteria and calculus. (200X).

An example of an inflammatory response in the muco-buccal fold, where a mass of organisms is caught in a crypt, is shown in Figures 10 and 11. This is added evidence of the injurious effects of the bacteria. The changes seen in the soft tissues are essentially the same as those described in the initial stages of periodontitis. As no tooth is present in this lesion to anchor the colonies of bacteria, the defense mechanism will soon dispose of the insecurely affixed microorganisms. With the outpouring of exfoliated epithelial cells, tissue fluids and pus, the injurious agents, temporarily entrapped within the crypt, will be washed away.

Sections cut horizontally at the neck of a monkey molar (Figs. 12 and 13), further clarify the manner in which tissues ad-

Fig. 11. High-power of crypt in Fig. 10. (465X).

jacent the enamel surface are affected by injurious agents. The cellular exudate is concentrated just under the epithelium next to the enamel with loss of the corresponding connective tissue fibers. Higher magnifications reveal passage of phagocytes through the epithelium to the tooth surface. Exfoliated epithelial cells are also found.

An extreme example of heightened activity within a periodontal lesion is displayed in Figure 14. This section is characteristic of a serial prepared from the upper right first molar of a 65 year old male who died with carcinoma of the esophagus. The lesion has its origin at the gingival crest. It progresses apically along the root into the bifurcation. It is within this deepest part that drainage occlusally is poorest. Consequently, all factors described previously as manifestations of periodontal disease are intensified at this level. They are illustrated in Figures 15 and 16.

DISCUSSION

"The life-cycle of the epidermal cells, which moves newly formed cells gradually outward, also causes any extraneous matter that enters the epidermis from below to be moved to the surface." It is within these words quoted from Rothman[22] that one finds a clue to the secrets of the cause and successful treatment of most periodontal disease. To them may be added the keen observations of G. V. Black,[23] "Within the subgingival space the layer of cells is much thinner, and especially are they much softer

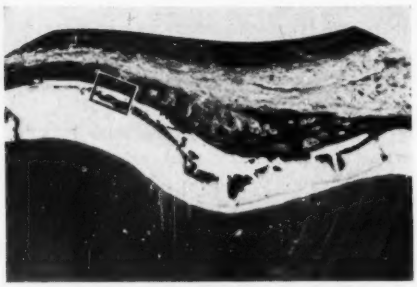

Fig. 12. Horizontal section, monkey molar. (12X).

and seem to permit, possibly facilitate, the escape of a fluid which continually bathes the subgingival space."

Mucous membrane has as one of its chief functions, absorption. It also secretes and is wet and slimy thereby aiding passage of various substances across its surface. Both clinical and laboratory studies provide substantial data that all of the gingiva is a form of mucous membrane with an active life cycle in accord with that of other mucous membranes in the body. The epithelial cells adjacent the tooth have all the properties of their brothers and sisters covering the alveolar surfaces. Their position next to the tooth provides them with

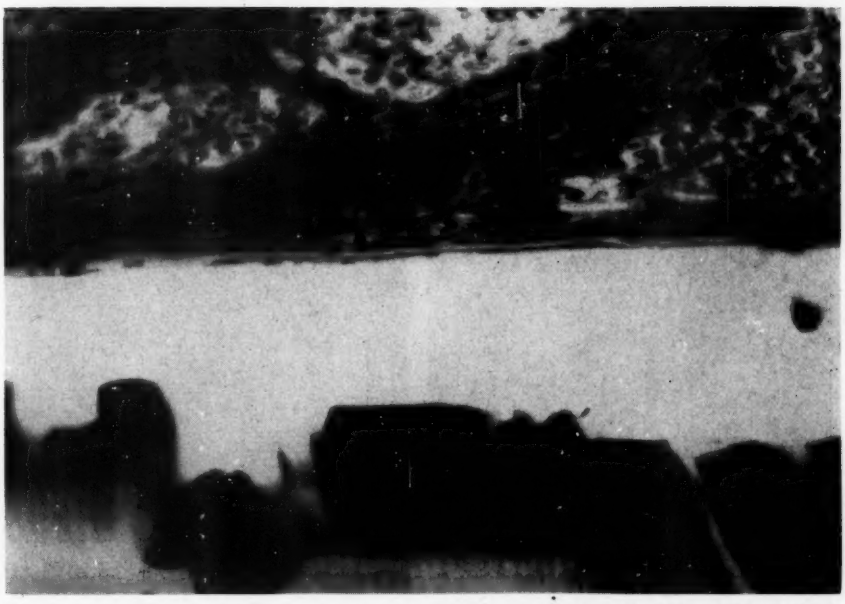

Fig. 13. High-power of rectangle, Fig. 12, with cellular exudate between enamel and epithelium (200X).

protection against many injurious agents, consequently, the layer is thinner, they are softer, they do facilitate the escape of fluids and cells, but proliferation and exfoliation are greatly minimized. Unfortunately, they apparently lose none of their absorptive function.

Fig. 14. Root bifurcation of upper molar, 65 year old male with advanced periodontal disease, P, pus. (12X).

With fixation of bacteria on the tooth surfaces next the gingival crest, toxins produced follow the line of least resistance between enamel and epithelium, and pass through the much thinner layers into the underlying connective tissues. This is the reason why the initial changes in gingivitis are seen next to the tooth surface. As the epidermal cells move from a proliferating basal layer outward, they are cast off. Those next to the tooth proliferate slowly in healthy gingiva. They also move outward, gradually working their way along the dental surfaces into the gingival sulcus and then into the mouth, providing they are not phagocytized or lysed en route.

The cells also produce a substance called "cuticle" that stains pink with eosin. This substance may be abundant when the

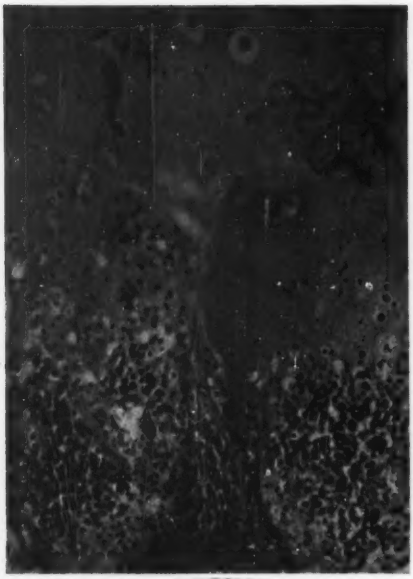

Fig. 15. Bottom of pocket, P in root bifurcation, Fig. 14. (200X).

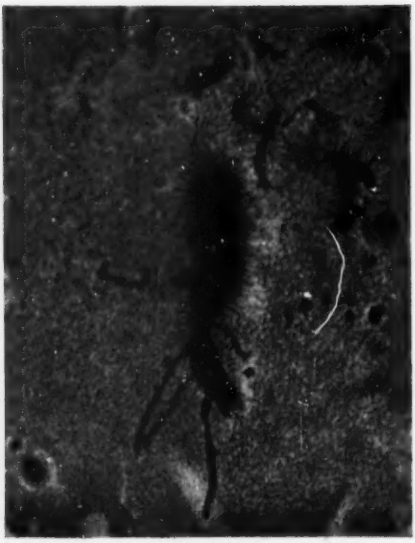

Fig. 16. High-power of microorganisms and pus seen in Fig. 15. (400X).

gingival epithelium is suffering from the effects of irritants. It is often difficult to find in strictly healthy specimens. It could be a secretion that, in its own way, assists in moving extraneous matter from below to the surface. It resembles, both in appearance and staining qualities, keratinous material found in tonsillar crypts. All agree the material in the crypt is soft and cheesy and that it is part of a detachment or exfoliative process. In spite of all these vital changes seen in the gingiva adjacent the enamel, the etiologic agents of periodontal disease find the "epithelial attachment route" to be the weakest link in the defense mechanism of the body. Consequently, the initial pathologic changes occur next to the tooth and not on the mouth surfaces of the gingival and alveolar mucosa.

Gingival health is a function of the two tissues, epidermal and connective. The epithelial cells play an active role in causing extraneous matter to be moved outward. As long as the connective tissue fibers are present in healthy abundance they maintain a close physiologic adaptation of the gingiva to the tooth surface.

SUMMARY

Careful analysis of the data presented arouses the following thoughts:

I. The Healthy Gingiva

A. Is physiologically adapted to the tooth surface by a dense fibrous body, containing little if any cellular exudate. Irritating bacterial clumps with calculus and toxins are present on the tooth adjacent the gingival crest in small amounts or not at all.

B. Is covered by epithelium that is thin adjacent the enamel and becomes thinner as the cemento-enamel junction is approached. Little evidence of proliferation is observed in the basal layers next to the tooth.

C. Responds to injury like other mucous membrane.

II. Periodontal Disease

A. Begins when injurious bacteria become affixed to and colonize on the tooth adjacent the gingival crest. Clumps of calculus form and germs grow freely on the surfaces and in the porosities thereof. Toxic products are produced.

B. Affects the living cells when the toxins are absorbed by the gingival tissues.

C. Is evidenced by epithelial proliferation, ingrowth, thickening, exfoliation and the usual inflammatory vascular response.

D. Leads to separation, degeneration and obliteration of the connective tissue fibers, thereby producing a swollen, flabby gingiva. A sulcus forms between the tip of the crest and the enamel.

E. Becomes well established as the bacteria follow the apical advance of the toxins along the enamel and root surfaces with additional calculus formation.

F. Develops into a serious clinical condition when soft tissue necrosis and ulceration occur. The massive fluid and cellular exudation, as well as exfoliation, provide decaying organic matter upon which the bacteria feed and multiply at an ever increasing rate.

G. Progresses to terminal stages as resorption of adjoining alveolar bone destroys tooth support.

The foregoing thoughts lead to the following hypotheses:

1. If no injurious bacteria were present, there would be no inflammatory reaction and consequently no lesion. The picture of the healthy gingiva in Figure 1 would be the rule rather than the exception.

2. The initial introduction of injurious agents between the enamel and gingival epithelium need not necessarily lead to a chronic lesion. If the injurious agent, corn husk, fishbone, or clump of bacteria, be dislodged before it is firmly attached to the tooth surface, physiologic readaptation could take place and there would be healing by resolution. It is only when injurious agents become firmly affixed that the disease process becomes chronic.

CONCLUSIONS

The conclusions that follow are derived from the foregoing hypotheses:

1. It is possible for the gingiva to readapt to the tooth surface, even though it has been disturbed by periodontal disease or treatment.

2. The vital anatomic, histologic and physiologic attributes of this tissue provide the dynamic power of response to injury that leads to healing by resolution or organization.

3. This satisfactory condition is achieved when all injurious agents are removed and the natural defense mechanism functions effectively.

4. Successful treatment of periodontal disease is based upon this knowledge.

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The Control of Bacteria in Gingival Pockets

by HOLMES T. KNIGHTON, D.D.S.,* *Richmond, Virginia*

IT is difficult to present anything sensational in a discussion on the control of bacteria in periodontal lesions. In fact, it seems rather generally agreed that: (1) bacteria play a secondary etiologic role in periodontal diseases, and (2) the bacteria involved are practically always of endogenous origin. In other words, bacteria involved in gingivitis and periodontitis are generally composed of an overgrowth of numerous species of the already existing oral flora. Furthermore, the initiation and retention of bacterial overgrowth depends upon various factors other than the bacteria themselves.

If the general and oral health is not impaired, the normal body defenses are efficient in keeping a proper balance between the oral flora and gingival tissues. Natural defenses include: the action of saliva, suction currents in the mouth, and the movement of the tongue, lips, and cheeks. The cleansing action of certain foods also aids in carrying bacteria from the mouth into the stomach. The intact gingival tissue including the attachment or physiologic adaption of epithelium to tooth structure acts as a mechanical barrier to bacterial invasion. There is also reason to believe that the normally existing oral flora exerts an antagonistic action on many chance contaminants that enter the mouth.

Oral hygiene measures including proper tooth brushing and proper use of dental floss supplement the natural defenses of the oral cavity. Such measures help to prevent the accumulation of food particles in gingival areas and in turn aid in preventing a bacterial overgrowth.

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If any one or combination of various factors interfere with the efficiency of normal body defenses and of the routine oral hygiene measures, then there may be an overgrowth of bacteria in gingival and periodontal areas.

Though bacteria very seldom initiate gingival and periodontal lesions, their overgrowth with their accompanying toxic and enzymatic products may account for an added irritating force.

Bacteria in periodontal lesions are included in an accumulation of debris, retained food particles, broken down tissues and cellular structure. This septic material may be protected from the normal body cleansing mechanisms and from routine oral hygiene measures by formation of calculus, deepened gingival sulci or periodontal pockets, malposed teeth, carious lesions, and poor dental restorations; that is, the organisms are no longer merely on the surface of epithelial cells.

Ordinarily discussions are in relation to the effect of bacteria on the lesions, but it should be remembered that the prevailing pathological conditions also influence the bacteria overgrowth.

The debris and necrotic material protect bacteria from body cellular defenses and from the quick germicidal action of chemical disinfectants. The extravasated blood, necrotic tissue and action of aerobic and microaerophilic bacteria combine to keep the oxidation-reduction potential suitable for a definite percentage increase of anaerobes, such as spirochetes, fusiform bacteria, vibrio and anaerobic cocci.

The following experimental evidence (Smith, 1949) based on studies of anaerobic infections in other parts of the body aids in explaining the percentage increase of anaerobes. Circulating blood has an Eh of from +126 to +246 MV which is considerably above +50 to +100 MV or the maximum allowing good growth of most anaerobes. Extravasated blood and anoxic tissue rather quickly drops to an Eh of +50 MV, well within the range of that allowing for anaerobic growth. The sulfhydral compounds in the broken down cellular structure aids in maintaining the reduced Eh. Thus conditions for the proliferation of both anaerobes and microaerophiles will continue in the septic areas until the debris, calculus and necrotic tissue are removed and a good blood supply returned.

REDUCTION OF BACTERIA

The most logical way to accomplish a lasting reduction of bacterial growth in periodontal lesions is to (1) correct the predisposing causes, and (2) cleanse the area by mechanical debridement. This does not remove all bacteria, but it lessens the number and, more important, it eradicates the nutritive and protective medium aiding bacterial growth.

Waerhaug's experiments (1952) present a good example of beneficial effects of cellular defenses if the debridement is adequate. If extensive pocket formation has occurred before debridement, then surgical removal of gingival tissue is the next consideration in preventing the reaccumulation of septic deposits.

In an appraisal of developments in periodontology from 1901 to 1950, Leonard (1951) stated that the tendency has been away from the use of drugs in the treatment of periodontal diseases. There is also much room for doubting the efficiency of local antiseptics such as quarternary ammonium compounds, organic mercurials, arsenicals, phenols, etc., in maintaining a low bacterial population in periodontal lesions.

In fact, there are many difficulties encountered in attempts to kill bacteria in periodontal lesions with chemical germicides. For example:

1. Debris and necrotic tissue protect bacteria from immediate contact with chemicals.

2. Organic material in the lesions has a neutralizing effect on many chemicals.

3. Chemical germicides that kill bacteria within minutes usually do so by coagulation of proteins. Such concentrations of chemicals able to reach and kill bacteria deep in protected areas would also cause destruction of surrounding tissue.

4. Diluting action of saliva tends to weaken chemicals from a germicidal range to mere bacteriostatic effects. Such bacteriostatic effects may then be neutralized before cellular defenses can aid in killing bacteria in the septic areas.

5. The catalase and reducing substances in periodontal lesions interfere with efficient germicidal action of oxidizing agents.

The difficulty of killing bacteria in and on tissues within a few minutes is illustrated by experimental results of Nungester and Kempf (1942). In brief, their experiments consisted of soaking a mouse tail in a broth culture of either pneumococci or *Streptococcus hemolyticus*. The tail was then allowed to dry and placed in a test tube containing a chemical disinfectant for two minutes, after which time a small section was snipped off and aseptically implanted into the animal's abdominal cavity. Cultures were made from heart blood of animals that died, before recording death as due to the test organism. Results in general showed that quarternary ammonium compounds and organic mercurials in recommended usable dilutions could not be counted upon to kill all the test bacteria in two minutes even on the surface epithelium.

Reasonable doubt of the ability of organic mercurials to render all streptococci and pneumococci non-virulent within 10 to 15 minutes is suggested by experiments of Morton, North, and Engley (1945). The latter investigators found that in numerous tests the organisms were capable of causing death of mice if injected after 10 to 15 minutes' direct exposure to the chemicals.

There is evidence that chemical antiseptics used locally do cause a temporary reduction of the oral flora as shown by tests with antiseptic mouth washes. In such tests counts are made of bacteria in saliva samples before and after use of antiseptics. It is not unusual in such tests to obtain an apparent 90 to 95 per cent reduction. These results simply indicate that there is a marked reduction of bacteria in the saliva and offer no direct evidence of numbers of bacteria left in the lesions. Let us assume, though, that this is an accurate estimate of viable bacteria remaining in periodontal lesions. If so, then the million or more bacteria found in each ml. of paraffin stimulated saliva, following the use of antiseptics, suggests that a significantly sizable inoculum is left for continued growth in the lesions. It seems logical then to agree with the opinions of numerous periodontists that local antiseptics are merely adjunctive agents in periodontal therapy. If such agents are used, the operator should not place undue confidence in their effectiveness to the extent of neglecting adequate debridement and correction of all probably etiological factors.

In the ever continuing search for specific or adjunctive therapeutic agents for gingivitis and periodontitis, it is obvious that sulfonamides and antibiotics would be given an extensive trial.

SULFONAMIDES

Sulfonamides gained some popularity in the late 1930's and early 1940's. The fact that these drugs were not particularly effective was indicated by Ostrander (1947) in a discussion of recent trends in the use of sulfonamides. The latter stated that sulfonamides have no place in the treatment of periodontal diseases and with the possible exception of acute cases have not been successful in treatment of Vincent's infections.

The partially broken down tissues, purulent exudate, and necrotic material in gingival lesions interfere with the action of sulfonamides due probably to both a

mechanical protective action and to the para-aminobenzoic acid present.

Lewis and Burket (1948) demonstrated a 31 per cent decrease in the oral flora 30 minutes after patients started chewing gum containing 0.25 grams of sulfathiazole. The reduction was 21 per cent after one hour, but the bacterial count rose to the original level one hour after discarding the gum. The authors suggested the need for chewing a fresh piece of sulfathiazole gum every 30 minutes for any prolonged depression of bacteria. The reported percentage reduction does not seem to be particularly significant in view of what can be obtained with antibiotics.

ANTIBIOTICS

Innumerable reports are available on the beneficial effects of antibiotics in treating oral lesions. There is still doubt, however, as to the need of antibiotics in gingival and periodontal lesions except possibly as an adjunct in treating acute necrotizing gingivitis.

Bacteriological data on effects of antibiotics on the oral flora are available, but are scant as compared with the numerous clinical reports. The following summarization indicates the possible effect that antibiotics may have on the oral flora.

Long (D.A.) (1947) reported an approximately 87 per cent reduction in the flora in saliva in from 1 to 2 days if 500 units penicillin pastiles were used every 45 minutes during the day or if 500,000 to 1,000,000 units were injected intramuscularly. There was also evidence of a reduction of fusiform bacteria and spirochetes as noted in smears in from 1 to 2 days with none or very few noted after 4 days. This report also mentions that within 2 to 7 days *C. albicans* and gram negative bacteria of the colon-aerogenes group grew in large numbers bringing the bacterial count back to its original high. There was a gradual return to the usual oral flora after discontinuance of penicillin. Bender and associates (1953) found no reduction in the oral flora 1 hour after injection of 300,000 units of procaine penicillin and 100,000 units of crystalline penicillin. Nor was there a reduction one hour after injecting 1 gram of dihydrostreptomycin. If, however, the penicillin and dihydrostreptomycin were injected together, there was a 65 per cent reduction within the hour.

Rosenthal (1950) noted a disappearance of motile spirochetes within 24 hours if 300,000 units of penicillin were injected intramuscularly or if 30,000 to 50,000 units of penicillin were used locally in the form of chewing troches.

Hutchinson and Low (1946) and Karlin and Crutchfield (1950) report significant reductions in fusiform bacteria and spirochetes in from 2 to 4 days, following use of penicillin troches. It is of interest to note that Hutchinson and Low found no evident effect with the penicillin therapy in 21 per cent of their 80 cases.

The relative ease of obtaining a temporary reduction in the oral flora by either local antibiotics or antiseptics is shown by Slanetz and Reynolds (1952). The latter report from 74 to 99 per cent reduction of bacteria in saliva samples within 5 days if troches containing either 500 units of penicillin or 1/1500 cepacol were used 30 minutes after each meal. Slanetz and Reynolds also mention a 43 to 92 per cent reduction within two hours after use of a 15 mg aureomycin troche. Kraus and associates (1951) present evidence of an approximately 86 per cent reduction of bacteria in saliva one hour after use of 15 mg troches of either aureomycin or terramycin. They also list evidence of significant reductions with systemic administration of these wide range antibiotics.

In preliminary work in our Medical College of Virginia laboratories, we have noted from 40 to 80 per cent reduction within 1 to 2 days following use of combined bacitracin (300 units), neomycin

(5 mg), and polymyxin (2000 units) in troches. The troches were used by subjects every two hours during the day. We have not been able to completely eradicate motile spirochetes deep in periodontal pockets by this treatment.

Since *Candida albicans* is probably present normally in from 25 to 33 per cent of mouths of humans, it is evident that there will be increased numbers in many individuals following antibiotic therapy. Slanetz and Reynolds (1952) and Carter and White (1953) offer some hope of partially controlling this overgrowth by combining certain quarternary ammonium compounds with penicillin in troches. More experimental work on both the need and efficiency of this procedure is needed.

Thus in summary it is possible to temporarily decrease the oral flora with a variety of antibiotics. If, however, the mouth is not debrided and predisposing causes of gingivitis and periodontitis corrected, the bacterial count usually returns to its original high after discontinuance of the antibiotic. The chief need for antibiotics in control of bacteria in these lesions seems to be: (1) as an aid in controlling the acute symptoms of necrotizing gingivitis and (2) in the control of bacteremias arising from extractions or scaling teeth.

If antibiotics were harmless, their routine use might be justified; but unfortunately, many people may become sensitized to the antibiotics and also bacterial strains may become resistant. Thus since there is so much evidence of satisfactorily controlling even the acute necrotizing gingivitis cases without antibiotics, it seems reasonable to question the need for their routine use.

Of course, the use of systemic administration of antibiotics as needed to control bacteremias in patients with heart lesions or certain other systemic diseases is an accepted procedure.

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The Reaction of Clinically Normal Gingiva to Abrasive Injury*

by JOHN A. KOLLAR, D.D.S.; FRANK M. WENTZ, D.D.S., PH.D.; BALINT ORBAN, M.D., D.D.S., *Chicago, Illinois*

DURING the course of series of experiments which were conducted to test the action of the air-abrasive technique upon both hard and soft tissues, observations were made that indicated a low inflammatory response and rapid repair of gingiva, which had been injured by the action of the air-abrasive spray.

The present investigation has been conducted in an attempt to clarify the gingival tissue changes incident to this type of superficial injury.

MATERIAL AND METHOD

Six subjects, including two of the authors, were used as experimental subjects. All individuals were free of papillary or marginal gingivitis. Three gingival papillae in each quadrant were abraded by using a coarse grit aluminum oxide powder with the Airdent machine. The gingival areas included the papillae between cuspids and first bicuspids; first bicuspids and second bicuspids; second bicuspids and first molars. A total of 12 gingival papillae were abraded in each patient.

Biopsies were taken before the experiment, immediately following injury; then, 6, 12, 18, 24, and 48 hours after abrasion. The first two biopsies were procured at 5:00 p.m., the following at 11:00 p.m., 5:00 a.m., 11:00 a.m., 5:00 p.m., and the last specimen was taken at 5:00 p.m. of the following day, 48 hours after the experimental injury.

The biopsies were procured under infiltration anesthesia with novocaine. The specimens were fixed Zenker Formalin solution, embedded in paraffin and stained with hematoxylin eosin after sectioning.

FINDINGS

Clinical Symptoms: The abraded gingiva appeared hemorrhagic and eroded. The surface appeared to be tattooed with the aluminum oxide (Fig. 1). Six hours after the injury the surface erosion was covered by an adherent crust-like material. Twenty-four hours after the injury the scab-like crusted surface could easily be detached and peeled away. After forty-eight hours no other sign of injury except an increased redness of the surface was apparent.

Figure 1. Clinical appearance of a gingival papilla immediately after injury by coarse grit, aluminum oxide, air-abrasive spray. The abraded surface appears eroded and hemorrhagic.

Subjective Symptoms: The air-abrasive action was carried out without novocaine. The subject experienced a burning sensation, momentarily, while the air-abrasive

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Presented to the General Meeting of the International Association for Dental Research, French Lick, March 19-24, 1954.

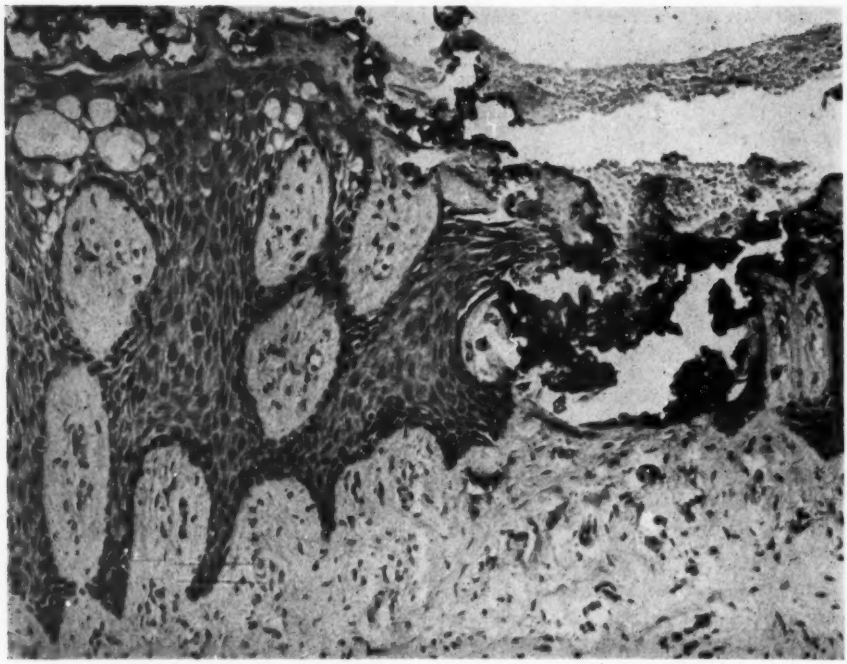

Fig. 2a. Photomicrograph of a gingival biopsy taken immediately after injury caused by air-abrasive spray. In some areas the suprapapillary epithelium has been completely destroyed.

spray was applied. A few minutes later the burning sensation disappeared and no spontaneous discomfort was experienced. However, during the next meal (2 hours later) the injured surfaces were extremely sensitive to chemical (vinegar) and physical irritation. Twelve hours after the experimental injury no painful sensation was experienced during eating.

Histologic Findings: Immediately after abrasion the injured area exhibited a freely bleeding surface. The biopsy specimen, taken at this time, showed that in some areas the suprapapillary epithelium was completely destroyed, exposing the connective tissue papillae while other areas showed epithelial remnants (Fig. 2a). Only in a few restricted areas was the injury deep enough to injure the epithelium of the interpapillary ridges. The irregularity of the damage was a very prominent feature in all biopsy specimens.

The surface of the injured area was covered by a hemorrhagic exudate incorporating a dark crystalline material and cell

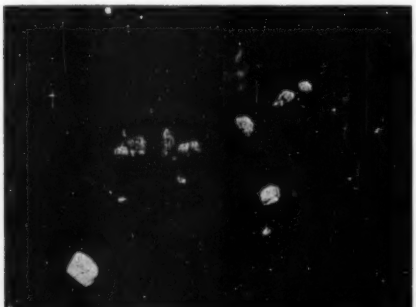

Fig. 2b. Photomicrograph of the same gingival biopsy as Fig. 2a using polaroid filters. The aluminum oxide crystals may be observed to be embedded in the epithelium and connective tissue.

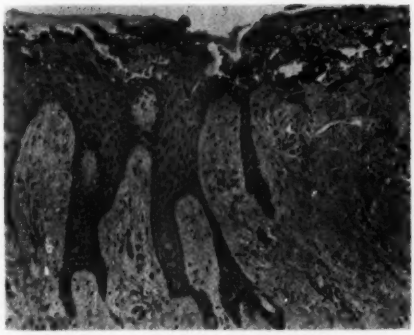

Fig. 3. Photomicrograph of a gingival biopsy taken six hours after injury by air-abrasive spray. There is little change from the earlier specimen. Signs of inflammation are slight and only a few polymorphonuclear leucocytes are observed.

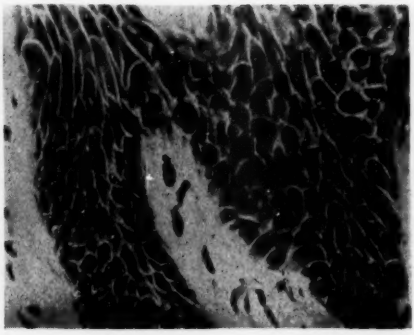

Fig. 5. Photomicrograph of epithelium of a gingival biopsy taken twenty-four hours after injury by air-abrasive spray. Observe that approximately five mitoses are occurring in the epithelial cell layer.

debris. The dark crystals were embedded in the epithelium as well as the connective tissue and may be best visualized with polaroid filters (Fig. 2b). The remnants of the suprapapillary epithelium showed extensive vacuolization.

In six hours not much change was observed (Fig. 3). The surface of the gingiva was still covered by the blood clot impregnated with the crystalline abrasive material and only the mildest signs of an inflammatory reaction could be observed; some of the blood vessels appeared slightly dilated and a few polymorphonuclear leucocytes were present in the connective tissue.

Six hours later and twelve hours after the experimental injury the histologic picture showed some changes. The surface clot was reduced. There was less crystalline foreign material. Epithelial migration over the denuded areas seemed to have begun. The connective tissue was slightly edematous and a few more leucocytes were present (Fig. 4).

Eighteen hours after the injury the wound surface was completely covered. The inflammatory reaction showed no increase in intensity and there appeared to be no sign of increased epithelial mitotic activity.

Clinically, at about 24 hours after abrasion most of the dark tattooed surface layer had peeled off. The epithelium covered the entire abraded surface and increased mitotic activity could be observed in the basal cell layers (Fig. 5). Only a few remnants of the necrotic tissue was visible on surface and the epithelium showed only a mild degree of edema.

The final specimen was taken forty-eight hours after the air-abrasive had been applied (Fig. 6). Now the only evidence of previous injury was the lack of epithelial ridges at the site of previous total epithelial

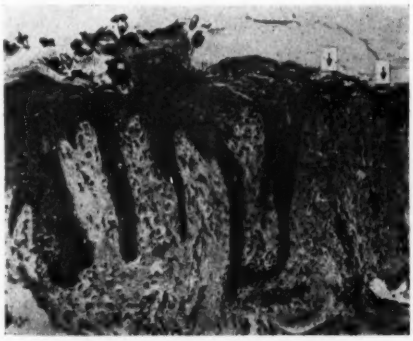

Fig. 4. Photomicrograph of a gingival biopsy taken twelve hours after injury by air-abrasive spray. Epithelial migration is beginning over the denuded areas (see arrows).

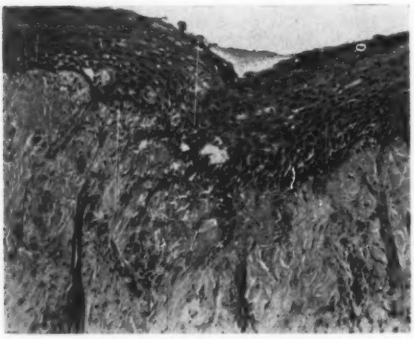

Fig. 6. Photomicrograph of a gingival biopsy taken forty-eight hours after injury by air-abrasive spray. The wound is completely covered by a thick layer of epithelium. The only evidence of previous injury is the lack of epithelial ridges and a few embedded crystals of aluminum oxide. Signs of inflammation are almost completely absent.

destruction and the presence of a few crystalline particles in the epithelium and connective tissue. The infiltration of inflammatory cells was slight.

Clinically, the area was slightly reddened but showed no evidence of erosion.

DISCUSSION

The interesting feature of this experiment is the rapid healing and almost complete lack of inflammatory reaction following mechanical injury. The absence of any but the mildest inflammatory reaction can be explained possibly by the superficiality of the injury, the lack of infection and the apparently innocuous character of the crystalline aluminum oxide.

As a rule repair is considered to be the outcome of an inflammatory reaction to an injury. The described experimental findings seem to show that there are exceptions to this rule under certain special conditions. In other words, one has to conceive the possibility that repair can follow immediately after injury. This conclusion tends to draw a sharper line between the two commonly correlated processes of inflammation and repair.

Confirmation of the observation that wound closure occurs not so much by mitotic processes but by migration of cells from the borders of the wound,[1,2] is afforded by the interesting finding that epithelial repair of the surface break was complete at about eighteen hours after injury before any increased mitotic activity was apparent. The latter was most prominent twenty-four hours after injury, but after the wound surface was well epithelialized.

SUMMARY AND CONCLUSIONS

The purpose of this investigation was to study gingival tissue changes that occur after injury by the air-abrasive spray.

1. The injury associated with the abrasive erosion is partial destruction of epithelium, damage of epithelium remnants, exposure of connective tissue, embedding of crystalline aluminum oxide in epithelium and connective tissue.

2. The inflammatory reaction associated with the injury is extremely mild, consisting of only vascular changes without a pronounced inflammatory cell infiltration.

3. Surface repair began at about twelve hours and was complete between twenty-four and forty-eight hours after injury.

4. Epithelial mitotic activity was most prominent at about twenty-four hours after injury.

5. The type of wound healing may be described as primary healing under a scab, Anderson.[2] The scab here is the blood clot impregnated with inert aluminum oxide crystals.

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DYNAMIC EQUILIBRIA OF ORAL TISSUES
II. Cytochrome Oxidase and Succinoxidase Activity of Oral Tissues

by PHILIP PERSON, D.D.S., PH.D., AND GEORGE W. BURNETT, D.D.S., PH.D.,
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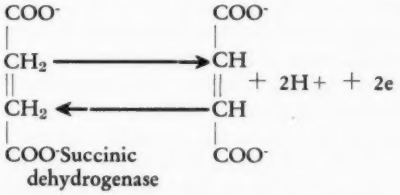
ALL aerobic microbial, higher plant, and animal cells possess within their mitochondria a system of iron-porphyrin-protein enzymes, known as the cytochromes.[1-3] By means of these catalysts, cells are able to utilize molecular oxygen for the complete oxidation of metabolites to carbon dioxide and water. The processes involved are those by which cells obtain energy for synthetic activities, for movement of ions against concentration gradients, for secretory activities, and for other endergonic processes which contribute to the dynamic equilibria of bodily constituents.

The essential feature of biological oxidations is a loss of electrons, and in Figure 1 are depicted the terminal stages of electron transport, effected by cytochrome enzymes. This vital system of respiratory enzymes can ultimately receive electrons from (i.e., oxidize) such major metabolites as carbohydrates, amino acids, fatty acids, and purine derivatives. The metabolism of these substances is channeled into the cytochrome system by a variety of metabolic paths,[4] only a few of which are indicated in the diagram.

Of particular interest in this series of enzymes is cytochrome oxidase, universally recognized as the major respiratory enzyme of mammalian tissues.[1,2] The function of cytochrome oxidase is to transport electrons between reduced cytochrome *c* and molecular oxygen within the cell. In other words, it is the catalyst which enables cells to utilize molecular oxygen. In the absence of cytochrome oxidase, molecular oxygen might remain in contact with many major biological substrates for years, without the occurrence of appreciable oxidation. Thus, in some unknown fashion, cytochrome oxidase "activates" molecular oxygen so that it will accept electrons more readily in biological systems. Synonyms for cytochrome oxidase are indophenol oxidase,[5] Atmungsferment,[6] saüerstoffübertragendes Ferment,[7] respiratory enzyme,[8] and oxygen activating enzyme.[9]

It has been known for many years that one metabolite, succinate, is far more active than any other naturally occurring substrate in restoring metabolic activity (as measured by oxygen uptake) to washed tissue preparations lacking endogenous substrates.[10] Succinic dehydrogenase, an enzyme of unknown composition is linked closely to cytochrome oxidase within cell mitochondria. It activates succinate to give up protons and electrons, thereby converting it to fumarate:



The electrons are transported via succinic dehydrogenase and ultimately arrive at cytochrome *c* via cytochrome *b* and at least one unknown factor.[11] From cytochrome *c* the final electron transfer to molecular oxygen is mediated by cytochrome oxidase. The entire system of enzymes involved in the aerobic oxidation

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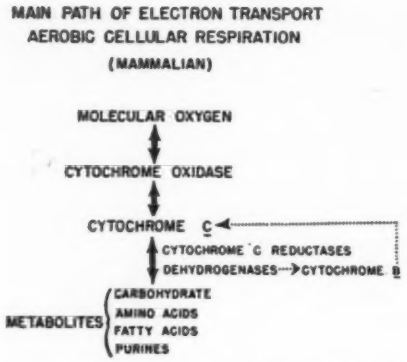

Fig. 1. A diagrammatic representation of the terminal stages of electron transport effected by the cytochrome enzymes during aerobic cellular respiration.

of succinate comprises succinic dehyrogenase, cytochrome *b*, an unknown factor, cytochrome *c*, and cytochrome oxidase; this complex is termed succinoxidase. It is easy to eliminate much confusion if one remembers that succinoxidase refers to a complex of enzymes, whereas succinic dehydrogenase refers to a single enzyme, responsible for the step in which succinate loses electrons and protons.

Previous studies have indicated that cellular metabolic responses to certain hormonal, drug, and nutritional influences may be mediated through the cytochrome and succinoxidase systems.[12-14] These enzymes have been investigated intensively elsewhere in the body,[3] but no studies have been made of their role in oral tissue metabolism. In this paper the results of the investigation of cytochrome oxidase and succinoxidase activity of rat tongue and submaxillary and parotid gland will be reported and compared with the activity of these enzymes in rat liver, heart, and kidney.

METHODS AND MATERIALS

The tissues utilized in this study were obtained from adult, male Sprague-Dawley rats weighing between 300 and 500 grams. The animals were raised on a diet of Purina laboratory chow supplemented with green vegetables. All animals were sacrificed by decapitation and bled. The organs were removed by dissection, chilled in cracked ice, blotted and weighed as rapidly as possible. They were quickly homogenized in cold ion-free water (prepared by passing distilled water through a mixed anion-cation exchange resin) at 4° C. in an ice-jacketed Waring Blendor for 45 seconds, then in a chilled, ground glass, Ten-Broeck tissue homogenizer. The tissue homogenates were finally diluted with water to the following volume per gram of wet tissue:

Tongue	35 ml
Parotid Gland	65 ml
Submaxillary Gland	35 ml
Liver	30 ml
Heart	65 ml
Kidney	65 ml

Cytochrome oxidase was determined manometrically in the Warburg respirometer at 37° C. using air as gas phase.[14] The system contained the following components in the Warburg flask: center well, 0.2 ml. of 10 per cent NaOH; side arm, 0.3 ml. of hydroquinone solution (= 3 mg. hydroquinone); body of flask, 1.0 ml. of $Na_2H\ PO_4$-$KH_2\ PO_4$ buffer (pH and molarity varied as described for each experiment), 0.1 ml. of cytochrome *c* (= 1 mg. of cytochrome *c*), 0.25 ml. of 0.1M semicarbazide hydrochloride (adjusted to pH 7.0 with NaOH), 1.0 ml. of tissue homogenate, and water to make final volume of 3.0 ml.

All reagents were added to the Warburg flasks immediately prior to preparation of homogenates. As soon as the latter were ready, they were pipetted into the flasks. When complete, the flasks were attached to open manometer and placed quickly into the constant temperature bath, where they were shaken at 120 times per minute for 10 minutes. The contents of the side arm were then tipped into the body of the flask. Shaking was continued for an additional five minutes after which the manometers were closed and the initial readings were made; final readings were made at the end

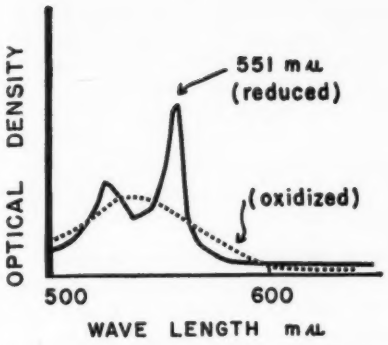

Fig. 2. The absorption spectrum of reduced and oxidized cytochrome c.

of 15 minutes. The values obtained were multiplied by four to convert them to oxygen uptake per hour. This procedure is valid since the oxygen uptake in the above system is a straight line function of time during the first hour.

The procedure for manometric assay of the succinoxidase complex is identical with that described above for cytochrome oxidase except for the reagents utilized. Reagents used in this system were: center well, 0.2 ml. of 10 per cent NaOH; side arm, 0.1 ml. of cytochrome *c* (= 1 mg. of cytochrome *c*); body of flask, 1.0 ml. of $Na_2H PO_4$-$KH_2 PO_4$ buffer (pH and molarity varied as described for each experiment), 0.3 ml. of 0.5M sodium succinate adjusted to pH 7.4; 0.3 ml. of a solution 4×10^{-3}M with respect to Ca++ and Al, +++ 1.0 ml. of tissue homogenate, and water to make a final volume of 3.0 ml. All determinations involving variables were made in duplicate; in the last two series of tests single determinations for cytochrome oxidase and succinoxidase activity were made on each tissue homogenate.

Nitrogen was determined by a Nessler procedure,[15] modified by the use of 20 per cent sodium citrate to prevent the formation of a precipitate when diluted homogenate digests were treated with Nessler reagent.

Cytochrome *c* utilized in this study was obtained from Wyeth and Company. The absorption spectra of the oxidized and reduced enzyme are shown in Fig. 2. Hydroquinone, C.P., was obtained from Amend Drug Company; sodium succinate, $6H_20$, was from Merck and Co. Ion-free water was prepared by passing distilled water through a mixed anion-cation exchange resin (Crystalab Deeminizer, Aloe Scientific Corp.). Buffer salts were reagent grade, Merck and Co.

EXPERIMENTAL RESULTS

Several variables may affect cytochrome oxidase and succinoxidase activity of oral tissues. The more important determinate variables affecting enzyme activity are: (1) method of preparation of tissue homogenate, (2) properties of substrates, (3) nature, pH, and concentration of buffer system, (4) temperature, and (5) ionic strength.

The method of preparing tissue homogenates has already been discussed, and it was found to be satisfactory for studying cytochrome oxidase and succinoxidase activity of oral tissues.

The substrates most commonly used for manometric determination of cytochrome oxidase activity are ascorbic acid, hydroquinone, and p-phenylenediamine, all of which are auto-oxidizable.[4] Hydroquinone was chosen as the substrate because it has been demonstrated that by adjustment of pH, its auto-oxidation in liver and heart homogenates, can be brought to negligible values or eliminated entirely.[14,16] The auto-oxidation of hydroquinone was reinvestigated, however, at varying pH and time intervals, in the presence of excess cytochrome *c*. The system was identical to that described under procedures, except that 1 ml. of water was substituted for 1 ml. of homogenate. The results are shown in Fig. 3. It may be seen that a pH value above 7, the auto-oxidation of hydroquinone is considerable, while below pH 7 it is slight. Blanks for auto-oxidation were included in

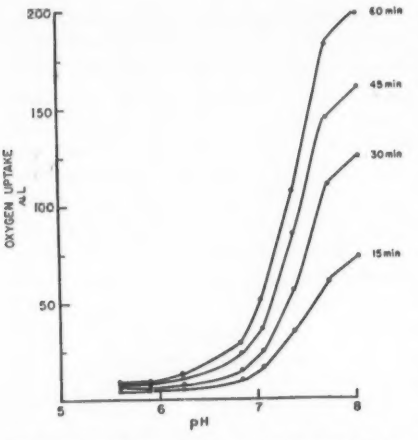

Fig. 3. The effect of pH on the auto-oxidation of hydroquinone at 37° C. in air, and in the presence of cytochrome c, (1 mg. per vessel).

all determinations, however, to eliminate the possibility of error from this source. Sodium succinate was not investigated in this fashion for it is not auto-oxidizable under conditions of these experiments.

Most studies of cytochrome oxidase and succinoxidase activity have used phosphate buffers,[4] and in previous studies, we have found them to be satisfactory.[3, 14, 16, 17] It was necessary, however, in this study to re-evaluate the interrelationship between buffer molarity and pH and their effect upon cytochrome oxidase and succinoxidase activity in the various tissues.

The effect of variation of pH on the cytochrome oxidase activity of homogenates of submaxillary and parotid glands, heart, kidney, tongue, and liver was first studied utilizing a O.1M Na_2H PO_4-KH_2 PO_4 buffer system in each case. The data which is compiled in Fig. 4 shows that each of the tissues had optimal enzyme activity between pH 6.8 and 7.1, with maximal activity near pH 7.1.

A fixed pH of 7.1 was then used to study the effect of variation in buffer concentration on the cytochrome oxidase activity of the tissue homogenates. The range of buffer molarity studied was between 0.05 and 0.4, at intervals of 0.05. From the data compiled in Fig. 5 it will be seen that at pH 7.1 cytochrome oxidase activity of each tissue homogenate was optimal between 0.1 and 0.3 molarity. However, the maximal enzyme activity of the tissues was near 0.2M, which was considered to be the working optimum.

Since the optimal pH for enzymatic activity had been determined in 0.1M buffer in the previous experiments, it was again necessary to ascertain whether the pH optimum would change when utilizing a 0.2M buffer. The cytochrome oxidase activity of submaxillary and parotid gland homogenates was determined in 0.2M buffer, with the pH ranging from 5.6 to 7.4, at five intervals. The data, compiled in Fig. 6, shows that enzymatic activity was optimal between pH 6.8 and 7.0.

The cytochrome oxidase activity of liver, kidney, heart, and tongue was also found to be optimal between pH 6.8 and 7.0. On the basis of the above data, it was concluded that conditions for cytochrome oxidase activity were optimal for liver, kidney, heart, submaxillary and parotid glands, and tongue homogenates at pH 6.9 and in a 0.2M Na_2H PO_4-KH_2 PO_4 buffer.

It was known from previous studies that succinoxidase activity in heart, liver, and

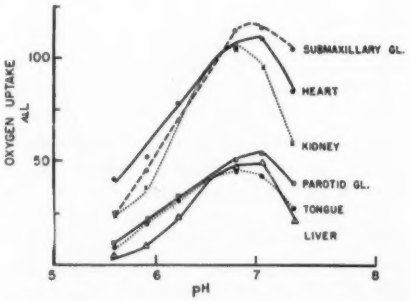

Fig. 4. The effect of pH on the cytochrome oxidase activity of various rat tissue homogenates in 0.1M phosphate buffer with air for gas phase, and at 37° C.

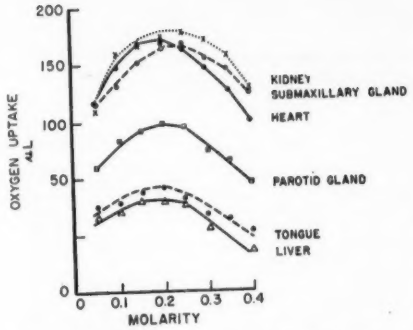

Fig. 5. The effect of phosphate buffer molarity on cytochrome oxidase activity of various rat tissue homogenates at pH 7.1, air as gas phase, and at 37° C.

kidney homogenates was optimal between pH 7.3 and pH 7.4.[14] To verify this finding the succinoxidase activity of submaxillary and parotid glands, liver, heart, tongue, and kidney was determined in a 0.2M $Na_2H PO_4$-$KH_2 PO_4$ buffer and at a pH range of 6.8 to 8.1, with measurements made at four intervals. The data compiled in Fig. 7 shows that enzymatic activity was near optimal at pH 7.4 for the tissue homogenates. The optimum buffer molarity for maximal succinoxidase activity of the tissue homogenates was then determined at pH 7.4. The molarity of the buffer solutions tested ranged between 0.05 and 0.35, at intervals of 0.05. The data in Fig. 8 indicates that optimal enzyme activity occurs

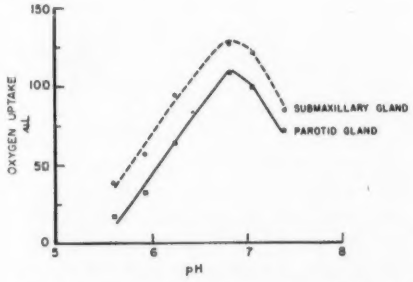

Fig. 6. The effect of pH on the cytochrome oxidase activity of rat submaxillary and parotid gland homogenates in 0.2M phosphate buffer, air as gas phase, and at 37° C.

with a buffer molarity of about 0.2. It was concluded, therefore, that pH 7.4 and 0.2M buffer concentration was optimal for testing succinoxidase activity in the various tissue homogenates.

As indicated in the literature most assays for cytochrome oxidase and succinoxidase activity have been made at 37° C., including those done in our previous studies. These studies have indicated that this temperature is suitable for measurement of such enzyme activity. The temperature of 37° C. was chosen for this study so that better comparisons may be made between the work now being presented and that already in the literature.

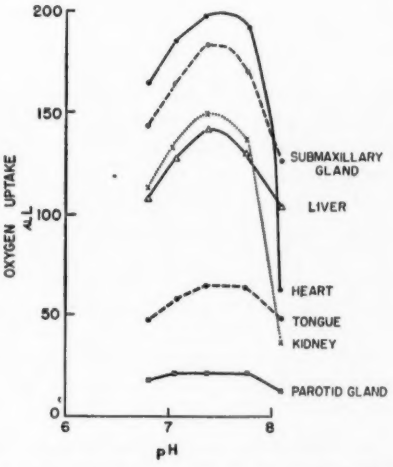

Fig. 7. The effect of pH on the succinoxidase activity of various rat tissue homogenates in 0.2M phosphate buffer, air as gas phase, and at 37° C.

In the next series of studies, the activity of cytochrome oxidase in homogenates of heart, kidney, parotid and submaxillary glands, liver, and tongue was determined at pH 6.9 and in 0.2M phosphate buffer. In these experiments, the various organs were obtained from seven adult, male animals, ranging in weight from 306 to 431 grams.

TABLE I

*Cytochrome Oxidase Activity of Tissue Homogenates of Sprague-Dawley Rats Expressed as Oxygen Uptake /μl/Hour/mg. Nitrogen**

Tissue	*Mean O_2 Uptake*	*Range of O_2 Uptake*	*SD*	*CV*	*SEM*
Heart	813	674-1004	103	12.7	39
Kidney	681	561-828	97	14.2	37
Parotid Gland	679	546-810	117	17.2	44
Submaxillary Gland	592	510-733	79	13.3	30
Liver	468	417-522	39	8.3	15
Tongue	375	230-505	86	22.9	33

*Individual observations were made upon organs removed from seven male rats weighing between 306 and 431 grams.

SD = Standard deviation; CV = Coefficient of Variation; SEM = Standard error of the mean.

One determination for enzyme activity was made on each homogenate. The data for cytochrome oxidase activity of these homogenates are compiled in Table I, showing the enzyme activity expressed as μl of oxygen consumed per hour per milligram of tissue nitrogen. It is to be remembered that the average error in the accuracy of manometric determinations of this type is about 10 per cent. It can be seen that the cytochrome oxidase activity of the tissues varies considerably, being greater in heart and least in tongue.

In Table II the succinoxidase activity is shown for the same tissue homogenates used in the previous experiment to determine cytochrome oxidase activity. The range of enzyme activity between different tissue homogenates is greater than that of cytochrome oxidase activity. Succinoxidase activity is greatest in heart and least in the parotid gland.

DISCUSSION

Homogenization of a tissue yields suspensions of the particulate components of protoplasm which include mitochondria, microsomes, secretory granules, nuclei, cell membranes, etc., together with freely diffusible components which include inorganic ions, enzymes, coenzymes, and other soluble materials. When properly prepared, sufficient dilution of such homogenates lowers the concentration of substrates and co-factors so that endogenous oxygen uptake is abolished or greatly reduced. It is then possible to study the reactions of a given enzyme in such homogenates to the exclusion of others, by addition of a suitable substrate and co-factors for only the given enzyme. By this method, it is often possible to define a particular reaction and to determine quantitatively the specific enzyme which catalyzes it.[18]

The homogenization technique is excellent for determination of some enzymes but not for others. Fortunately, it is suitable for studying cytochrome oxidase and succinoxidase activity, for it has been established that the reactions occurring within tissue homogenates with respect to these enzymes also occur within intact tissues.[1, 2, 8] Therefore, while the use of homogenates to study cytochrome oxidase and succinoxidase activity is arbitrary to some extent, it is also well-founded. The data obtained by the use of the isolated enzyme systems in homogenates will prove valuable in studying the more complex reactions of intact tissue systems. It is our intention to extend this investigation in the future to include the cytochrome oxi-

TABLE II

*Succinoxidase Activity of Tissue Homogenates of Sprague-Dawley Rats Expressed as Oxygen Uptake /μl/Hour/mg. Nitrogen**

Tissue	*Mean O_2 Uptake*	*Range of O_2 Uptake*	*SD*	*CV*	*SEM*
Heart	1256	1107-1361	98	7.8	37
Kidney	983	812-1032	93	9.5	35
Submaxillary Gland	549	340-646	96	17.5	37
Liver	462	364-547	68	14.7	26
Tongue	426	362-464	47	11.0	18
Parotid Gland	217	192-290	33	15.2	12

*Individual observations were made upon organs removed from seven male rats weighing between 306 and 431 grams.

SD = Standard deviation; CV = Coefficient of Variation; SEM = Standard error of the mean.

dase and succinoxidase activity of tissue slices and intact tissues.

In general the effects of pH and buffer molarity upon cytochrome oxidase and succinoxidase activity are similar to those already described in the literature.[17] Previous studies have indicated that a phosphate buffer molarity of 0.1 at pH 7.1 was suitable for the assay of cytochrome oxidase and 0.1M at pH 7.4 for succinoxidase activity. It was fortunate, however, that the variables affecting the activity of these enzymes were re-examined in this investigation. Under present conditions cytochrome oxidase activity was maximal in 0.2M phosphate buffer at pH 6.9, while succinoxidase activity was maximal in a similar buffer at pH 7.4. While this may appear to represent a small variation in the influence of pH and buffer molarity on cytochrome oxidase activity, it is significant nonetheless, considering the auto-oxidizability of hydroquinone.

Since the enzymes of the various tissues behaved similarly with respect to pH and buffer concentration optima, it is presumptive evidence that they may be similar in each tissue. This assumption does not mean that factors regulating enzyme activity are the same in all tissues, however, for it must be recognized that regulation of enzyme activity may be as important to the economy of the cell as is the existence of the enzyme.

The variation of the succinoxidase activity curves (Fig. 8) in response to varying buffer concentration raises an interesting point concerning interpretation of these data. It is known that succinoxidase is a very labile enzyme system and undergoes

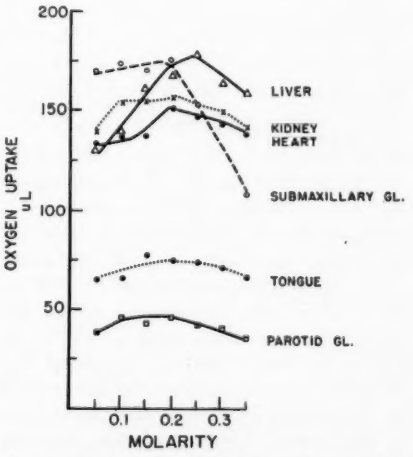

Fig. 8. The effect of phosphate buffer molarity on various rat tissue homogenates at pH 7.4, air as gas phase, and at 37° C.

rapid denaturation. If such lability is the cause of the variations of succinoxidase activity, a similar response might be expected when varying the pH of the tissue homogenates. Since this did not occur, we must look elsewhere for an explanation of the irregularity of succinoxidase activity. It may be that such irregularities are caused by differences in cellular compositions which are reflected as differences in colloidal behavior.

Both pH and buffer molarity affect ionic strength, an important factor in the physico-chemical behavior of all proteins.[19] Satisfactory control of ionic strength is easiest in purified enzyme preparations. While the task of accurately computing ionic strength in tissue homogenates of varying composition is not insurmountable, it was not considered wise to do so under conditions of the present experiments, for data concerning contributions of the tissues themselves to ionic strength are not available. Previous attempts by Wainio, Person, Eichel, and Cooperstein to establish a relationship between ionic strength and activity of heart muscle cytochrome oxidase, of much greater purity then the present homogenates, were unsuccessful.[17] It is known that such anions as Cl^- inhibit cytochrome oxidase and succinoxidase,[20] but the only exogenous source of Cl^- in these experiments is from the semicarbazide-hydrochloride used as quinone fixative. The magnitude of the contribution from this source (final concentration 3×10^{-6}M Cl^-) is negligible.

The data in Tables I and II demonstrate that the oral tissues exhibit marked oxidative activity and indicates that they are significant participants in the dynamic equilibria of bodily constituents. While the range of activity of the enzymes in the tissue homogenates are indicated, it is not wise to make conclusions concerning the observed orders of activity, for they would be meaningless at this time. The importance of these results lies in their demonstration of the significant oxidative metabolism occurring within oral tissues. The magnitude of the oxidative metabolism of oral tissues is equal to that of the other organs studied, which are considered to be major metabolic sites. The extent to which oral tissues participate in the dynamic equilibria of bodily constituents will be put to subsequent experimental tests.

SUMMARY

1. The cytochrome oxidase and succinoxidase activities of tongue, parotid and submaxillary gland have been studied and compared to those of heart, liver, and kidney of adult, male Sprague-Dawley rats.

2. Optimal pH and buffer concentration, using Na_2HPO_4-KH_2PO_4 buffer, were determined at 37° C. for enzymatic activity of tissue homogenates prepared in ion-free water.

3. The oral tissues studied possessed significant aerobic oxidative metabolic activity.

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Pathogenesis of Pocket Formation in Traumatic Occlusion*

by JENS WAERHAUG, D.D.S., PH.D., *Oslo, Norway*

A HEALTHY gingival crevice is one in which normal epithelial cells join the tooth surface from the deepest point of the epithelial cuff (epithelial attachment) to the gingival margin.[41] After the eruption of the tooth the bottom of the crevice is, under favorable conditions, to be found at the cemento-enamel junction.[41] An apical migration of the crevice beyond the cemento-enamel junction means a loss of periodontal fibers and must be considered a pathological condition.[41]

"Traumatic occlusion" is defined by Stillman[36] as "a condition where injury results to the supporting structures by the act of bringing the jaws into closed position." Karolyi[20] seems to have been the first to call attention to occlusal stress as an etiological factor in pyorrhea. His conception about etiology, symptoms and treatment was mainly the same as that held by present day authors which support this theory. The majority of publications [1, 4, 7, 9, 11, 12, 16, 17, 20, 23, 24, 25-27, 33, 35-37, 40] on traumatic occlusion deal with the clinical aspect and most of the authors consider traumatic occlusion to be an important causative factor which alone can produce pocket formation. Some authors maintain that occlusal trauma acts only as a secondary factor.[11, 13]

The effect of traumatic occlusion on the supporting structures has also been evaluated on the basis of histological investigations of autopsy material.[2, 3, 6, 10, 18, 19, 21, 28, 29, 31] The most common finding was circulatory disturbances. Some authors observed necrosis of the periodontal membrane on the pressure side,[3, 10, 15, 18, 28, 29] and pieces of cementum torn loose from the dentin on the stretch side.[2, 10] All the reports concerning tissue reaction to occlusal trauma in man are limited to the reaction in the periodontal membrane within the bony alveolus. No evidence seems to have been given indicating that circulatory

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disturbances and necrosis can be produced in the area between the alveolar crest and the deepest point of the epithelial cuff or the gingival margin. Thus Grohs[18] states: "No connection between marginal inflammation and the changes in the periodontal membrane could be found."

The pathogenesis of pocket deepening is discussed by Box[6] in the following words: "The gingival third of the pericementum . . . . is a peculiarly vulnerable region. Its proximity to the gingival crevice with its bacteria ever ready to invade the gingival tissues and establish infection, and its epithelial attachment inherently predisposed to cover the detached soft tissues by inward growth along the cementum, makes it especially liable to involvement by these processes." Box has not shown photomicrographs which demonstrate this procedure of pocket deepening.

A number of authors[5, 8, 14, 30, 32, 34, 38, 39] have studied the effect of excessive occlusal stress in animal experiments. The most extensive of these works was done by Gottlieb and Orban[14] on dogs. On the pressure side they observed necrosis of the periodontal membrane, abscess formation and rear resorption of the alveolar bone. Only in exceptional cases did they find downgrowth of epithelium below the cementoenamel junction in young animals, and if it occurred it was found only on the tension side. This downgrowth they considered to be caused by food impaction and not by trauma. In old dogs they found more extensive pocket formation and destruction of the alveolar bone. In cases where the crowns causing the trauma had been removed some time before the animals were sacrificed they generally found complete healing of the periodontal membrane and no permanent damage done. A clear conclusion of this work is expressed by Gottlieb[15] in these words: "As long as the destruction takes place at a distance from the epithelial attachment the immediate danger is not great. Repair may take place at any future time. But if the destruction occurs in the neighborhood of the epithelial attachment, the denuded root surface may be overgrown by epithelium, and the periodontal membrane loss is permanent." Gottlieb and Orban[14] drew the conclusion from their experiments that occlusal trauma does not play the role as a primary factor in pyorrhea. Similar conclusions also are drawn by others.[8, 13, 30, 34, 39]

In contrast to the previously mentioned authors, Box[5] and Stones[38] found more pocket deepening and downgrowth of epithelium on experimental teeth than on control teeth. Stones' conclusion is that: "Traumatic occlusion is a predisposing factor in the production of periodontal disease, but not the only cause."

As may be seen from this survey of the literature, opinions are widely divergent concerning the effect of occlusal overload on the destruction of the supporting tissues of the tooth. Therefore more facts are necessary before a definite opinion can be formed on this problem. The present investigation was started with the purpose of finding out to what extent longstanding repeated occlusal overload would lead to a deepening of the pocket.

MATERIAL AND METHOD

Seven dogs were used for the experiment, five of them being littermates. The experiment was started at an age of about eight months in six of the animals. The age of the seventh dog was unknown. Two of the dogs contracted distemper shortly after the experiment was started and had to be sacrificed. They were at that time in very poor physical condition. The occlusal overload was produced by means of a high crown on the lower right first molar occluding with the upper fourth premolar. In dogs the cusp of this upper tooth reaches some millimeters below the gingival margin on the buccal surface of the lower tooth (Fig. 1). As the intention was to produce a combination of vertical and horizontal overload, the type of crown illustrated in Figure 2 was used. The crown has a shelf on the buccal surface into which the cusp of the antagonist is biting. The

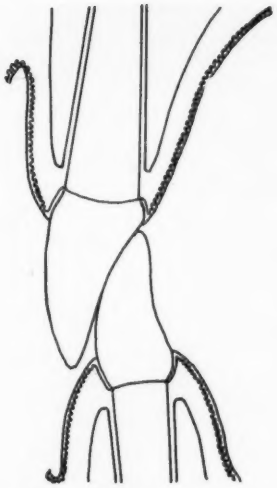

Fig. 1. Approximate relationship between lower first molar and upper fourth premolar in a dog when jaws are in occlusion.

direction of the pressure is apical and buccal in the lower jaw, and apical and lingual in the upper jaw. Thus the bite was raised approximately seven millimeters. In order to increase the retention, a series of holes was made near the margin of the crown and, after cementation of the crown, these holes were extended into the tooth surface and filled with amalgam. This procedure gave a solid fixation.

In three of the dogs the crown was left in place until the end of the experimental period. These are called short term experimental animals. In the remaining four animals, which in the text are mentioned as long term animals, the crown usually was left in place until the two antagonists were so much intruded that normal occlusion was re-established on the other teeth. The crowns were then removed and the teeth were allowed to extrude until normal occlusal contact was restored between the two experimental teeth. The crowns were then put on again. In a number of cases the crowns fell off because of vigorous chewing, for which reason chance to a certain degree has decided the periods in which the crowns were in place. As one of the purposes with the long term experiment was to find to what extent healing will occur when trauma is relieved, the crowns were removed some time before the animals were sacrificed.

The dogs were fed an adequate diet with ad libitum supply of bones for gnawing. After observation periods varying from 23 to 875 days they were sacrificed and the jaws fixed in formalin. The tooth carrying the crown and its antagonist, as well as the lower left first molar were selected for sectioning. The specimens were embedded, some in celloidin and some in paraffin, cut in buccolingual direction and stained with hematoxylineosin.

OBSERVATIONS

The animals were examined regularly during the observation period. After the crowns had been put on, the animals chewed nervously and irritatedly. However, as the teeth intruded, the animals became accustomed to the crowns and the nervous

Fig. 2. Bite raised by means of a high crown on the lower first molar. Crown has a shelf on buccal surface onto which upper fourth molar is biting. Direction of force apical and buccal in lower jaw and apical and lingual in upper jaw. Additional retention provided by means of amalgam locks anchored in cavities in tooth.

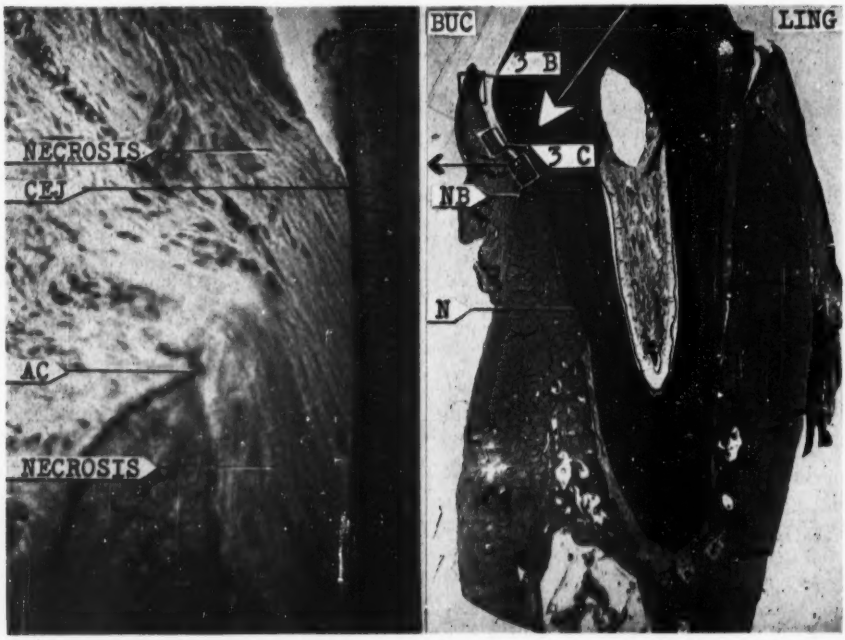

Fig. 3A. Dog 10. Distemper during observation period. High crown applied 23 days before death. Direction of force apical and buccal. Periodontal membrane in marginal third is wide on tension side and narrow on pressure side. In apical third, width of periodontal membrane reversed. Tooth intruded and periodontal membrane necrotized from N to alveolar crest. Formation of new bone, NB, on outside of alveolus corresponding to necrotic periodontal membrane. Higher magnification of area between alv. crest and CEJ seen to the left. Necrosis of periodontal membrane within alveolus extends a certain distance above cemento-enamel junction, CEJ. Only slight inflammation in connective tissue adjacent to necrotic area. No downgrowth of epithelium below cemento-enamel junction.

chewing decreased. When the crowns were in position, the lower first molars became very mobile, and when pressure was applied the animals showed obvious signs of pain. The crowns facilitated retention of food along the gingival margin.

The histological pictures which will be shown are all from the lower first molar. Figure 3A shows the results of a short term experiment in one of the dogs which contracted distemper. The crown was in place at the time of death. The observation period was 23 days. In the marginal area the periodontal membrane is wide on the lingual side and narrow on the buccal side. In the apical area these conditions are reversed. On the lingual side the alveolar crest reaches to the level of the cemento-enamel junction. On the buccal side the alveolar crest ends 0.2 mm below the cemento-enamel junction, and a broad rim of new bone, NB, is seen on the outside of the alveolus. The periodontal membrane is necrotized from N to a little above the alveolar crest. A higher magnification of the area between the cemento-enamel junction, CEJ, and the alveolar crest, AC, is shown to the left. The periodontal membrane is necrotized all the way to the cemento-enamel junction and even the lower part of the epithelial cuff just above the cemento-enamel junction is necrotic. In Figure 3B is shown a higher magnification from the gingival margin. Between the bacterial plaque and the epithelium is a thick layer

Fig. 3B Fig. 3C

Fig. 3B. Higher magnification from gingival margin in Fig. 3A illustrating marginal gingivitis caused by bacterial plaque.

Fig. 3C. Higher magnification from area between gingival margin and cemento-enamel junction in Fig. 3A illustrating lack of continuity between marginal inflammation and traumatic lesion.

of pus cells, consisting mainly of polymorphonuclear leukocytes. In the subjacent connective tissue there is a slight chronic inflammation. In Figure 3C there is a higher magnification from the area between the gingival margin and the cemento-enamel junction. The inflammatory signs are here minute.

Conditions are rather similar in Figure 4 which is taken from the other dog with distemper, the observation period being 42 days. The tooth is intruded and moved buccally. The periodontal membrane is necrotized in a short area below the alveolar crest on the pressure (buccal) side. Higher magnifications from the apical and coronal borders of the necrotized area are shown to the right. Osteoclastic resorption takes place in the alveolar bone both above and below the necrotized area. To the left there is a higher magnification from the area between the alveolar crest and the cemento-enamel junction. In the cementum there are Howship's lacunae containing osteoclasts. The inflammatory reaction in the connective tissue is slight. The distance from the alveolar crest to the cemento-enamel junction is 1.5 mm.

Figure 5 is taken from a tooth which had worn a crown alternately for 215 days (+ 58 — 40 + 72 — 45), it being 45 days since the crown was removed for the last time. There is formation of new bone, NB, in the upper wall of the mandibular canal. The width of the periodontal membrane is normal. In a higher magnification to the right it may be seen that a downgrowth of epithelium of 1.0 mm has taken place below the cemento-enamel junction. In the connective tissue there are signs of a slight chronic inflammation.

The experiment of the longest duration is illustrated in Figure 6. The crown had here been worn alternately for 875 days (+ 57 — 41 + 107 — 46 + 95 — 73 + 117 — 25 + 115 — 199). A period of 199 days elapsed after the crown was removed for the last time. There is normal width of

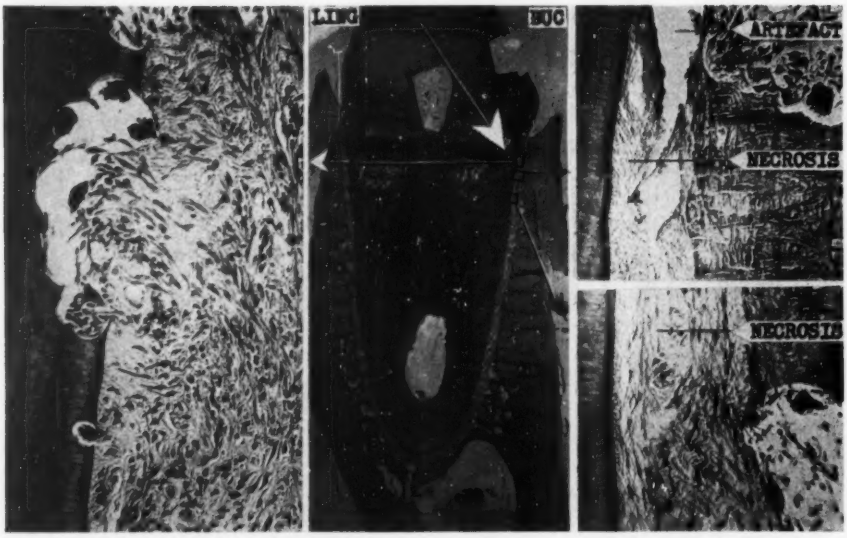

Fig. 4. Dog 9. Distemper during observation period. High crown applied 42 days before death. Higher magnification to the right shows upper and lower border of necrotic periodontal membrane on pressure side. Undermining osteoclastic resorption going on. Necrosis limited to periodontal membrane. Resorption of cementum and dentin halfway between alveolar crest and CEJ (see higher magnification to the left). Inconsiderable inflammation adjacent to necrotic area. No downgrowth of epithelium below CEJ.

the periodontal membrane and a solid alveolar wall. Below the cemento-enamel junction on the buccal side there are deep resorption areas in the dentin in which new cementum has been deposited (see higher magnification to the right). There is no downgrowth of epithelium below the cemento-enamel junction. On the lingual side the root surface has been resorbed near the apex and new cementum formed in the resorption lacunae.

Figure 6 is representative of the healing as it occurred in all the long term experiments with the exception of LR9 in Dog No. 2 which is shown in Figure 5. Resorption of cementum had not taken place to the cemento-enamel junction in all the cases, but there was always found some resorption somewhere in the marginal third on the pressure side. In Table 1 the presence (+) or absence (—) of resorption lacunae on the buccal and lingual third of the two experimental teeth and of the control tooth is indicated. The distance from the alveolar crest to the deepest point of the epithelial cuff has also been measured and tabulated. Cementum resorptions were found on the pressure side in all the long time experimental animals. On the stretch side they were found only in one case, and in the control teeth they were not observed at all. The distance from the alveolar crest to the epithelial cuff was found to be close to 1 mm, there being hardly any significant difference between the experimental teeth and the control teeth.

DISCUSSION

The most striking feature in the short term experiments is the necrosis of the periodontal membrane in the marginal third on the pressure side (Figs. 3 and 4). The necrosis may even reach into the principal fibers above the alveolar crest (Fig. 3), and here we are seemingly dealing with the salient point in the pathogenesis of pocket deepening in traumatic occlusion. As long as the necrosis does not reach all the way

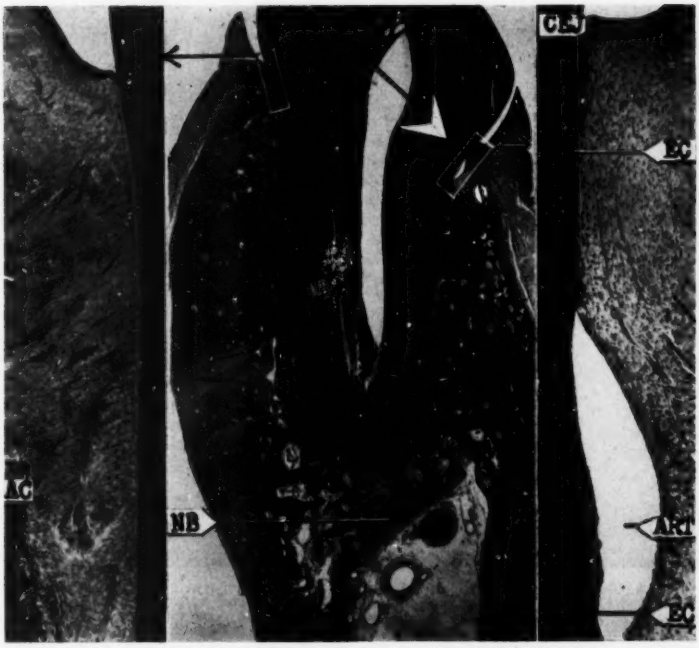

Fig. 5. Dog 2. Total observation period 215 days and crown worn alternately: +58 −40 +72 −45 days. Tooth has been intruded into alveolar canal, but extruded again during the last 45 days after crown was removed. Formation of new bone, NB, in alveolar canal. Normal height of alveolar crest on lingual side. On buccal side downgrowth of epithelial cuff to EC, 1 mm below CEJ, see higher magnification to the right. ART, artefact.

to the cemento-enamel junction no downgrowth of epithelium can come about, as it is stopped by healthy Sharpey's fibers attached to the cementum. If all these fibers are necrotized or completely broken down, there is no barrier to prevent downgrowth of the epithelium. Downgrowth of vital epithelial cells cannot, however, take place into the necrotic area, since, as illustrated in Figure 3, the same trauma that caused the death of the connective tissue cells will cause the death of the epithelial cells. Downgrowth of epithelium must, consequently, take place during a healing period after the necrotic tissue has been resorbed and before new principal fibers have been formed to prevent further downgrowth. In the short term experiments (Figs. 3 and 4) only a slight inflammatory reaction can be seen in the tissue directly adjacent to the necrotic areas, which indicates that the necrosis is aseptic. This slight inflammation shows no tendency to spreading and has no connection with the inflammation in the gingival margin, which is caused by bacterial plaque. This lack of inflammation in the area between the traumatic lesion and the marginal inflammation is well illustrated in Figure 3C. It is not very likely, therefore, that a *marginal gingivitis* can be caused by a traumatic overload on the teeth. This finding is in accordance with observations made by Grohs.[18]

The average distance from the alveolar crest to the cemento-enamel junction in the four long term animals where the high crowns had been removed some time before death, was about 1.0 mm (Table 1), and this figure is very near to what was observed on all the other teeth in these animals. In Figure 3 the corresponding

Fig. 6. Dog 5. Total observation period 875 days. High crown worn alternately: +57 −41 +107 −46 +95 −73 +117 −25 +115 −199. Normal height of alveolar crest and normal width of periodontal membrane. Deep resorption of cementum all the way to cemento-enamel junction, see higher magnification to the right. Resorbed area healed with secondary cementum into which are fastened normal Sharpey's fibers. No downgrowth of epithelium below cemento-enamel junction. Resorption in apical area shown in higher magnification to the left.

distance is, however, only 0.2 mm. The heavy overload on this tooth has caused an intrusion which has developed so rapidly that the alveolar crest has not been resorbed with the same speed. The alveolar crest has therefore touched the tooth so near to the deepest point of the epithelial cuff that all the principal fibers and all the cementoblasts have been necrotized. That is most likely what has happened in Figure 5, where a pocket deepening of 1.0 mm has occurred. In Figure 4 there also has been a considerable intrusion of the tooth, but the alveolar crest here has been resorbed with greater speed—the distance between the alveolar crest and the cemento-enamel junction being 1.5 mm. The possibility must, however, be considered that earlier in the observation period the conditions have been much the same as in Figure 3. A great bulk of the alveolus adjacent to the necrotic periodontal membrane may have been removed by undermining resorption, and such resorption is still going on adjacent to the remaining necrotic area. Whatever the sequence of events has been, there has been no downgrowth of epithelium in Figure 4. Immediately below the cemento-enamel junction healthy periodontal fibers are attached to the cementum and inflammation is absent. The reactive changes have, however, resulted in resorption of the cementum and the dentin. Resorption of cementum and dentin is, contrarily to the downgrowth of epithelium, not a permanent damage, and healing may take place in the form of deposition of secondary cementum in which are inserted Sharpey's fibers. This is what happened in Figure 6 and it is remarkable that in this case the cementum has been resorbed all the way to the cemento-enamel junction without any downgrowth of epithelium as a consequence. As is apparent from Figure 4, all the Sharpey's fibers have to be broken down before resorption can take place.

Figure 6 demonstrates, consequently, that pocket deepening need not necessarily take place even if all the Sharpey's fibers below the deepest margin of the epithelial cuff are destroyed. Osteoclastic resorption and cementum formation requires active participation of the cells involved, and this may act antagonistically to proliferation of epithelial cells. Whatever the mechanism may be, nature seems to be well prepared to prevent permanent damage in the form of downgrowth of epithelium.

In the long term experimental animals (Table 1) it may be seen that the distance from the cemento-enamel junction to the alveolar crest is about the same on the experimental teeth as on the control teeth. Clinical observations showed that the teeth were deeply intruded when the crowns were in place and this intrusion is demonstrated histologically in Figures 3 and 4. When the crowns were removed the teeth extruded again in a comparatively short time until normal occlusion was re-established. This means that the alveolar crest has been re-built as the tooth extruded, and it is remarkable that the distance to the epithelial cuff is about the same as in the control teeth. There seems, thus, to be a certain factor which determines that the alveolar bone cannot exist nearer to the deepest margin of the epithelial cuff than a certain distance, and in dog this distance seems to be about 1 mm. On the other hand there seems to be a growth factor which starts a formation of bone around the tooth if the distance to the deepest point of the epithelium is greater than about 1 mm. Impossible to explain as this phenomenon may be, it seems to be one of the basic protective mechanisms in the supporting structures of the teeth.

It is usually agreed that the supporting structures of the teeth are little suited to withstand horizontal stress, and that a strictly horizontal force causes more damage than does a strictly vertical force. There is reason to believe that such a statement lacks foundation. A strictly horizontal force may compress the periodontal membrane and cause necrosis, but an aseptic necrosis does not seem to cause any mentionable inflammation in the surrounding tissue; and if the alveolar crest is as far as about 1 mm from the cemento-enamel junction there will most likely not be destruction of the periodontal fibers to such an extent that downgrowth of epithelium can occur. A sudden horizontal overload combined with a certain amount of vertical stress may, on the other hand, cause an intrusion of the tooth to such an extent that the alveolar crest meets the tooth in the area of the deepest point of the epithelial cuff, and then conditions which open the possibility of downgrowth of epithelium are established. According to this way of thinking the supporting structures of the teeth are well suited to prevent permanent damage caused by horizontal trauma. They may even withstand horizontal trauma better than vertical trauma.

The area of principal fibers (approx. 1 mm wide) between the alveolar crest and the deepest line of the epithelial cuff represents a safety zone. This band of tissue follows the movements of the tooth and will not be injured by the same trauma that causes injury to the periodontal membrane within the alveolus. Before pocket deepening can be started, the traumatic inflammation has to reach the epithelial cuff, but as is apparent from Figures 3 and 4 there is very little inflammation caused in the principal fibers by the sterile necrosis in the periodontal membrane within the alveolus.

In the four long term experimental animals the high crown was put on and removed two to five times during the observation period, and the experimental teeth were not allowed to adjust themselves in any position for any length of time during that period. This was believed to be as unfavorable a set of conditions as it is possible to produce in an artificial way. The dogs, for their part, contributed to this end by chewing vigorously on the high crowns. To what extent this form of occlusal overload is comparable to

TABLE 1

In this table are tabulated the presence (+) or absence (−) of resorption lacunae in cementum and the distance from alveolar crest to deepest point of epithelial cuff in marginal third on tooth carrying high crown, LR9, and its antagonist, UR8, as well as control tooth, LL9.

Dog No.	LR9 carrying high crown.				UR8 antagonist to tooth carrying high crown.				LL9 control tooth comparable to tooth with crown.				Observation period in days. + indicates no. of days high crown was in place. − indicates no. of days high crown was removed.
	(pressure) Buccally		(stretch) Lingually		(stretch) Buccally		(pressure) Lingually		Buccally		Lingually		
	Resorption of tooth in marginal third	Distance alv. crest to epithelial cuff in millimeter	Resorption of tooth in marginal third	Distance alv. crest to epithelial cuff in millimeter	Resorption of tooth in marginal third	Distance alv. crest to epithelial cuff in millimeter	Resorption of tooth in marginal third	Distance alv. crest to epithelial cuff in millimeter	Resorption of tooth in marginal third	Distance alv. crest to epithelial cuff in millimeter	Resorption of tooth in marginal third	Distance alv. crest to epithelial cuff in millimeter	
	Short term experiments. Crown in situ when animal was sacrificed.												
4	7+	0.5	−	0.5	−	1.2	+	1.5	−	0.8	−	0.7	+97
9	−	0.2	−	0.0	−	0.8	−	0.9	−	1.1	−	1.1	+28
10	+	1.5	−	1.0	+	1.0	−	0.6	−	1.2	−	1.0	+42
	Aver-age	0.7		0.5		1.0		1.0		1.0		0.9	
	Long term experiments. Crown removed some time before animal was sacrificed.												
1	+	0.8	−	0.8	−	1.0	+	1.8	−	1.3	−	1.0	+58−40+135−34=267
2	+	0.6	+	0.8	−	1.6	+	0.9	−	0.8	−	1.0	+58−40+72−45=215
3	+	0.9	−	0.8	−	1.1	+	1.3	−	1.1	−	1.0	+58−42+137−15+19−27 +50−30=377
5	+	1.1	−	1.0	−	0.3	+	1.2	−	1.1	−	1.1	+57−41+107−46+95−73 +117−25+115−199=875
	Aver-age	0.85		0.85		1.0		1.3		1.1		1.0	

"traumatic occlusion" in man cannot be decided with certainty, as human material is not available; but it may be supposed that trauma in dogs is much more vigorous. An opening of the bite of 7 mm on one single tooth in each jaw will hardly ever be done in man. The premature contact which often has been made responsible for pocket deepening in man usually cannot be counted in millimeters but in tenths of a millimeter; and excursions of that magnitude will hardly be able to produce the kind of reaction which was observed in Figures 3 and 4. So even if it has been demonstrated (Fig. 5) that pocket deepening can be produced on teeth exposed to extreme trauma which intrudes them several millimeters, it is not allowed to conclude that the same pathogenesis can be applied when comparatively small "premature contacts" are concerned.

The dogs from which sections are shown in Figures 3 and 4 were in extremely poor physical condition. To what extent this has affected the present picture cannot be decided, but it is not likely that the main features of inflammation and healing have been interfered with to any great extent.

Traumatic occlusion is a term usually used by clinicians. "Traumatic" indicates that the tissue has been or can be injured, but in most cases that can hardly be decided by a clinical examination. It would, therefore, be advisable to use a more unspecific term like undue occlusal stress or occlusal overload.

CONCLUSIONS

1. Under extremely unfavorable conditions a deepening of the clinical pocket below the cemento-enamel junction can be produced by undue occlusal stress.

2. A combination of horizontal and vertical overload may cause an intrusion of the tooth to such an extent that it touches the alveolar crest in the area of the deepest point of the epithelial cuff. In that way the periodontal membrane may be necrotized all the way to the epithelial cuff and no Sharpey's fibers are present to prevent downgrowth of epithelial cells.

3. Downgrowth of epithelium can hardly take place as long as the periodontal membrane is necrotic, but must occur during a healing period after the necrotic tissue is resorbed and before new periodontal fibers have been formed.

4. A sterile necrosis within the periodontal membrane causes little inflammation, and it is hardly likely to produce an inflammation in the gingival margin.

5. The 1 mm wide area between the alveolar crest and the deepest line of the epithelial cuff represents a safety zone which follows the movement of the tooth and is not injured by occlusal overload. This zone prevents damage, done to the periodontal membrane within the alveolus, from spreading to the marginal gingival.

6. Damage done to cementum, periodontal membrane, and alveolar bone will be repaired when the tooth readjusts itself in a new position.

7. Downgrowth of epithelium is permanent damage.

8. The supporting structures seem to be well suited to prevent permanent damage caused by occlusal overload.

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Oral Orthopedics—A Concept of Occlusion

by the GROUP FOR ORTHOPEDIC RESEARCH IN DENTISTRY*

FOR many years periodontists have been concerned with the problems of occlusion and during this time have developed varying concepts regarding traumatic and atraumatic occlusal relations. Oral orthopedics concepts broaden the scope and definition of occlusion with special reference to the extra-oral as well as intra-oral factors which influence occlusal status. Thus the dentist's diagnostic horizon is enlarged, and new technics for achieving optimal results in periodontal treatment are provided.

Oral orthopedics also has an important place in the field of dental medicine because of the assistance it offers, for example, in the diagnosis and relief of certain pain and other syndromes of the head and neck.

Dentists have had to solve many problems in their efforts to bring their health services to their present high level. One of the earliest problems was securing correct functional relationship between the upper and lower artificial teeth in full denture construction. At that time there was no concept of mandibulo-maxillary relations or of balanced occlusion in the modern sense. The squash bite was used as a means of determining the relations between the arches; results were highly uncertain and unsatisfactory.

Studying the problem under the stress of frequent failures, dentists came to the conclusion that the answer lay in finding an anatomical landmark which could be used as a reference point in determining existing jaw relations. The condyle-fossa unit seemed to meet this need and study of that structure led investigators, further, to believe that the condyle was the controlling factor in determining the position and excursion paths of the mandible. Acceptance of these assumptions, which now seem not to be entirely valid, has had an unfortunate tendency to make inquiry into other factors seem unnecessary, thus hampering needed advance.

Work based on the assumed regulatory role of the condyle led to the invention of the anatomical articulator and the face bow. With the aid of these, the search for a concept of balanced occlusion was accelerated, still, however, using the condyle as the only point around which actions relating to occlusion centered.

THE PERIODONTAL PROBLEM

At a later time it was realized that traumatic occlusion was an important factor in the etiology of periodontal disease from both the therapeutic and preventive standpoints. The search for improved methods of achieving balanced occlusion gained new impetus, and the procedures involved came to be used in partial dentures, fixed and removable, as well as full denture prosthesis.

Studies of balanced occlusion as related to periodontal problems were carried out simply with the objective of obtaining non-injurious tooth-to-tooth relations. However, because of complications arising from retrusion, prognathism or other improper jaw relations, it often was found that intra-oral balancing alone did not suffice for the attainment of lasting success in treatment. In some cases orthodontics seemed to offer a satisfactory solution. However, traditional orthodontic procedures often failed to fully resolve the problem.

Attention was called by Victor Stoll[1] to other factors. His investigations brought out the fact that where jaw relations are incorrect there is an accompanying muscle imbalance—hypertonicity of some muscles and, often, hypotonicity of their opposites. These imbalances result in misdirected oc-

*The group consists of Drs. George D. Kudler, Robert Ritt, John Oppie McCall, Myron M. Lieb, Arthur A. Friend and J. Edward Curry.

clusal stresses with attendant traumatic occlusion. Stoll's researches thus broadened the concept of balanced occlusion to include the factors of jaw and muscle balance in addition to tooth-to-tooth relations.

Local therapy is of course essential in periodontal treatment. But important as such treatment is and excellent as are the results thus achieved, these results often are not lasting even in the absence of complicating systemic factors if occlusal faults have not been fully corrected.

JAW MALPOSTURE AND PAIN SYNDROMES

Some years ago Costen[2] called attention to the fact that certain symptoms felt in the ear and the region of the temporomandibular joint often had a dental cause. His view was that the symptoms were due to pressure on the auriculo-temporal nerve produced by displacement of the condyle in the fossa. Dentists already were interested in condyle-fossa relations, and some had come to the conclusion that pain and other syndromes felt in that region were due to condyle malrelations. Costen's claims of actual pressure on the nerve have been refuted, but he is to be credited with emphasizing the fact that disturbances in the condyle-fossa region may have a possible dental origin. Unfortunately his work tended to confirm the opinion held by dentists that it was pressure of the condyle in the fossa which caused the various syndromes associated with his name. However, Stoll and others studying the problem of mandibular muscle imbalance came to the conclusion that factors other than condyle pressure were responsible for the symptoms described. (The possibility that arthritic or other pathologic conditions of the joint may occur is recognized but incidence of such cases is low.)

STOLL'S CONTRIBUTION

In studies begun about thirty-five years ago, Stoll made a searching analysis of the fundamentals of jaw relations and the bearing of improper jaw relations on oral and general health. He found that in many cases deviations from normal functional pattern may bring about conditions which heretofore have been regarded as structural abnormalities. Examples are: retrusions, prognathisms, rotations and the like. These postural aberrations may develop as a result of faulty habit, malocclusion, tooth extraction or other disturbing factors. Patients with improper posture of the mandible of whatever origin present abnormal tonicity of the musculature and this has been found to play an important part in the etiology of many intra- and extraoral disturbances.

With regard to pain syndromes of the head and neck, it has been found that these frequently are referred pains originating in strains of the musculature, associated ligaments and blood vessels of the part. Such strain is most frequently caused by jaw imbalance, faulty masticating habits, faulty patterns of swallowing, respiration and speech; and other factors.

A wide variety of symptoms, some consisting of pain, others manifested in disturbances of function as, for example, certain types of hearing loss, are often associated with jaw malposture. Facial pain simulating tic douloureux, burning tongue, also occipital pain, often accompanied by stiffness of the muscles of the back of the neck, have been traced to jaw malposture. This has been true also of certain types of headaches[3] and pains radiating to the shoulder girdle.

BODY MECHANICS

Correct jaw posture is a prerequisite for proper balance of the head on the spine. And this, in turn, is needed for correct body posture and balance, a requisite for body comfort and health.[4] Conversely, poor body posture may act as a cause or contributing factor in faulty jaw posture.

According to Stoll, "When a part of the body, e.g., the mandible, assumes an abnormal position in relation to the rest of the body, it upsets the correct postural maintenance of the body against gravity.

And in order to maintain equilibrium and balance it calls on one set of muscles to function at the expense of others. It puts a strain on those muscles and creates in them a state of increased tension and in the opposing muscles a state of decreased tension. By upsetting structural relationships and neuromuscular balance it may cause various symptoms such as muscle spasm, distant referred pain and direct injury secondary to the disturbed posture."

DENTAL MEDICINE

Many of the disturbances traceable to jaw malposture are outside what has in the past been regarded as the dentist's field. But the fact that their occurrence may have a dental background and thus require dental attention for their alleviation projects the dentist into what has heretofore been a dento-medical no man's land. The oral orthopedic approach provides the knowledge and technics required. Because of its systemic as well as dental relationships the oral orthopedic concept broadens the scope of dental medicine.

THE DENTAL APPARATUS

Dentists have given attention chiefly to structures which they consider as constituting the masticatory mechanism—namely the teeth and their supporting tissues, the jaws, the muscles of mastication and the temporomandibular joints. It has not been sufficiently appreciated that mastication, deglutition, respiration and speech bring into action not only the jaws and their immediately attached muscles but also the muscles of the tongue and cheeks and other related muscles and associated structures of the head, neck and shoulder girdle. All these structures must be considered in studies of oral function and are included under the term "dental apparatus."

The intimate role of the hyoid mechanism in the functions of the dental apparatus, of which it is an integral part, has been consistently underestimated. The hyoid bone, lying just beneath the mandible, is in the form of the letter U and on a small scale resembles the mandible. The mandible, the hyoid bone and the anterior part of the shoulder girdle form a series of bow-shaped structures with interconnecting musculature. The hyoid bone forms a sort of scaffold against which the supra- and infrahyoid muscles act. The interconnecting musculature of these bow-shaped structures, together with the supramandibular musculature, form a suspensory apparatus which controls mandibular function. Taken together, these muscles also have an important function in maintaining the balance of the head on the spine.

The structures of the neck include, in addition to the hyoid mechanism, the larynx, the trachea and the thyroid and cricoid cartilages with their muscular attachments and fascial sheaths. These, together, form a funnel-like arrangement between the mouth and the thorax. It has been demonstrated that any compression of this composite structure caused, for example, by improper posture of the mandible, will affect the functions of swallowing, breathing and speech, with adverse effects also on aural ventilation and hearing.

FACTORS WHICH CONTROL MANDIBULAR POSTURE

The mechanism which controls the posture and the functional movements of the mandible is primarily its musculature; the condyle plays a secondary role in these functions. Stoll stated, "It is absolutely erroneous to assume that the condyle *altogether* guides the movements of the mandible and that the condyle has a definite position in the fossa which is fixed at birth." This has been confirmed by others, as, for example, Nove[5] and Haupl.[6] Other researchers in dentistry today are becoming aware of the fact that the musculature of the mandible plays a prominent part in its posture and excursions, a concept which accordingly minimizes the role of the condyle. However, the profession at large is still preoccupied with the matter of condyle-fossa relations and this attitude

conditions their approach to studies of occlusion and jaw relations.

METHODS OF SECURING JAW REGISTRATION

As already noted, study of methods of determining jaw relations involves the consideration of bite registration and the mounting of models. With the advent of the anatomical articulator came efforts to devise registration procedures which presumably would make it possible to avoid errors so often occurring with the older squash bite. The Gothic Arch tracing, developed many years ago by Gysi, has been widely used. McCollum has advocated the concept of the hinge axis in securing correct jaw registrations. Throughout the development of these technics runs the idea that when the condyles are in or are brought into their "centric" position in their fossae, the mandible will be in its correct position.

Stoll, realizing that emphasis on condyle relations was wrongly placed, was more interested in being able to analyze possible discrepancies in the relations of the mandible to the maxilla and of the jaws to the head. The postural status of the mandible, considered as a component of body posture, is to be determined in relation to the midsagittal, horizontal and transverse planes of the head. Stoll's researches demonstrated that when the original relationship is faulty in any respect correction of the malposition of the mandible can and should be made. He devised an instrument which aids in the analysis of the relationship of the mandible to the maxilla. Where a malrelationship exists the instrument helps in determining the correction needed.

REST POSITION

Many dentists regard rest position as a reliable index of ideal mandibulo-maxillary relations and thus useful as a base in planning correct occlusal relations of the teeth or their substitutes. In rest position the jaws are parted slightly, thus providing what is called the free way space.

According to the proponents of rest position technics, the criterion for correct relationship of the jaws is the attainment of correct position of the condyles in their fossae. The effort to achieve this is stressed particularly in cases of patients with disturbances believed to have their source in malposition of the condyles.

The basis of the rest position technic is the principle of muscle balance. In this respect the rest position approach and the principles of oral orthopedics are in agreement. There are, however, important points of disagreement.

Some patients have assymmetrical muscle tensions which tend to make it difficult for them to attain the state of muscle balance on which the success of the rest position technic is predicated. For those patients some dentists[7] prescribe exercises whose object is to secure the relaxation of tense muscles and thus permit bringing the mandible into its correct position. But in cases in which there are muscle imbalances of long standing, experience has shown that such exercises alone may not give the desired results.

There is another point to be considered in the matter of securing optimal jaw relations. There is evidence that rest position changes under certain conditions, contrary to accepted opinion. Walsh[8] has shown that the concept of an unchangeable rest position is not physiologically tenable. In support of that claim he cites studies of patients who had lost posterior teeth and thus had experienced a change in incisal guidance due to resulting loss of vertical dimension. In those cases he found a change in rest position and stated that such changes may be brought about by alterations of the reflex neuromuscular mechanisms governing both mastication and jaw posture. In the light of the discovery that such changes are possible it is evident that the patient's present rest position obtained by customary methods may not be the optimal position.

In cases in which growth and develop-

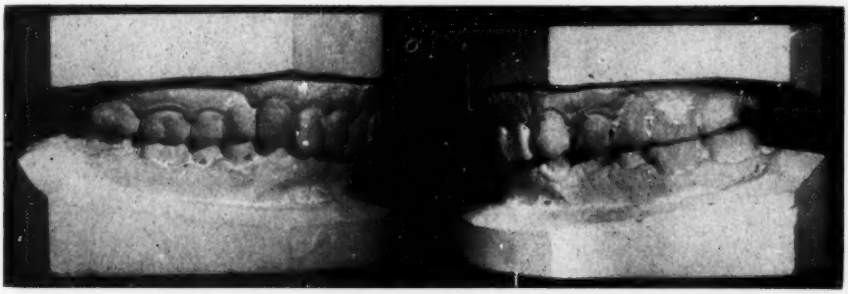

Fig. 1. Case 1. Models showing relations before treatment. Note extreme overbite of anterior teeth.

ment have been normal and where there is no abnormal muscle tension and no abnormal conditioned reflex, correct rest position may be obtained by technics customarily practiced. However, rest position technics or other commonly accepted technics for jaw registration, when used in cases having abnormalities such as those indicated, are likely to perpetuate the imbalances, with continuance and acceleration of periodontal pathologic processes.

In the cases reported below jaw imbalance was present, accompanied by muscle imbalance, such imbalances having a decidedly prejudicial effect on periodontal health.

REPOSITIONING THE MANDIBLE

Basic among the requirements for normal occlusion, using this phrase in its broadest sense, are normality of structure and normality of function. Structural normality involves, among other factors, consideration of tooth-to-tooth relations, relations of the jaws to each other and to the head, and the structural aspects of the musculature. Functional normality rests on balanced action of the muscles in the multiple functions they perform. This requires, among other things, the correction of perverted habits if present.

Repositioning the mandible when indicated is being practiced by a growing number of dentists. Lindblom[9] has discussed this at length. But for him, as for proponents of the rest position technic, the objective is to bring the condyles into what is believed to be their ideal position in their fossae. Oral orthopedics procedures are based on the principle that balancing the mandible and its musculature in relation to the other structures of the head and neck is the prime end to be sought and that the position of the condyles in the fossae is of secondary importance.

CONSIDERATIONS OF TREATMENT

The first step in treatment is diagnostic —the determination whether jaw malfunction and/or malposture is present. The procedure includes comprehensive case history, clinical examination, including consideration of the status of the musculature, an analysis of jaw relations with the use of study models, and other aids. Roentgenograms include the usual intraoral series, temporomandibular and lateral head-and-neck films. The relationship of the jaws to each other is best studied with the use of the specially designed Stoll analyzer.

Treatment involves a consideration of both structure and function. In the structural phase study is made of the occluding surfaces. Prematurities if any are noted and corrected and a wax bite in the corrected relationship is formed and tried in the mouth. When the patient accepts the relation set up by the trial bite, such steps as are needed to provide for occlusal interdigi-

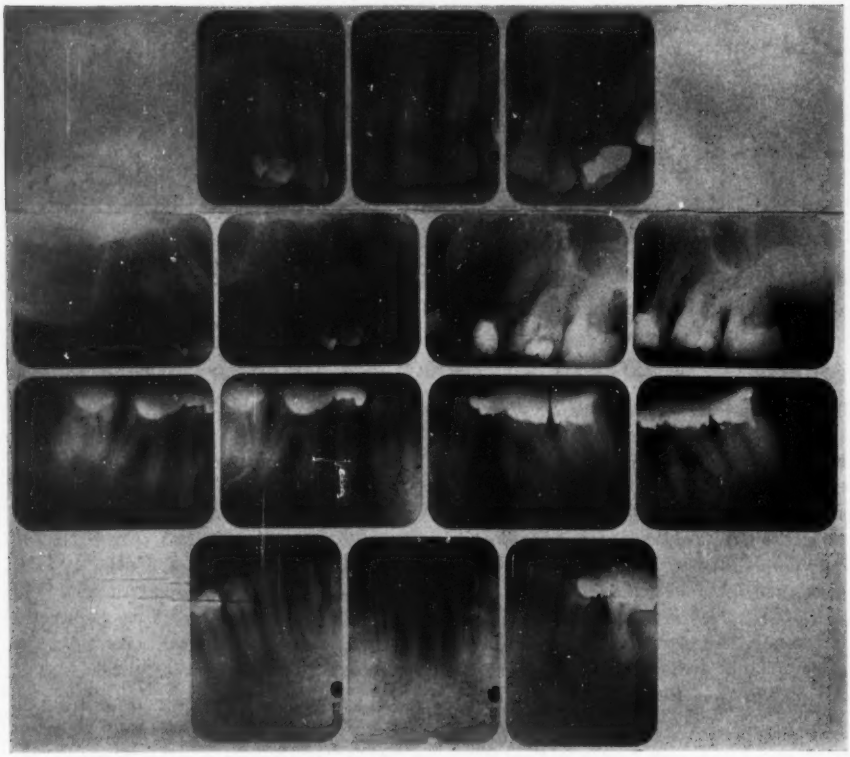

Fig. 2. Case 1. Roentgenograms of case before treatment.

tation in the corrected relationship are taken. In many cases temporary or permanent splints are constructed.[10] Frequently elimination of prematurities will suffice.

Along with the structural phase of treatment a functional phase is instituted. Muscle re-education is begun. Myofunctional exercises are used to establish correct conditioned reflexes which will guide the mandible into the desired position. Elimination of faulty habit patterns if present is requisite. With the structural assistance described the establishment of a correct functional pattern is facilitated and at the same time the patient is given a feeling of security on closing in the new position.

Some reflex patterns have a proprioceptive origin and are functionally involuntary. Others can be brought under voluntary control. Changes in the latter may be developed through repetition of prescribed exercises; through such repetition these voluntarily controlled actions become both unconscious and involuntary. McLeod[11] stated that normally "mastication is an unconditioned reflex; that is, it occurs automatically as a result of appropriate stimulation. However, modification of the conditions under which these inborn responses occur may be brought about as a result of experience or training, and these responses to new forms of stimuli are called conditioned reflexes."

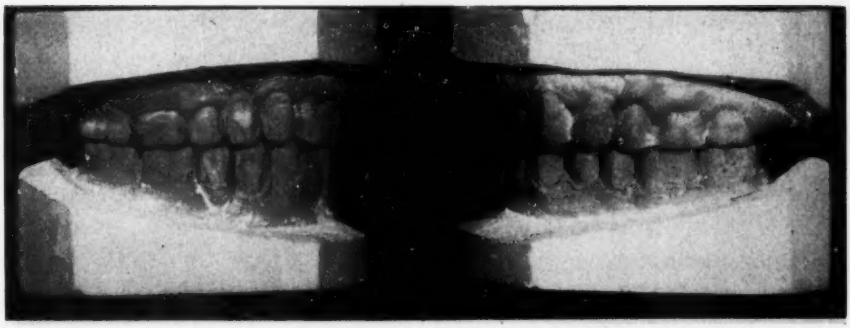

Fig. 3. Case 1. Models showing relations after repositioning. Note reduction of overbite and forward repositioning of the mandible.

CONDYLE POSTURE IN JAW REPOSITIONING

When the position of the mandible is changed to even a slight degree there will be some change in the position of the condyle. Ordinarily this is slight in extent and may not register measurably in the roentgenogram. In other cases the extent of change may be noticeable. It has been the opinion of the profession that it is not permissible to reposition the mandible if that involves bringing the condyle noticeably out of its centric position. That such repositioning can properly be done, however, is evidenced by the anatomical fact that, contrary to earlier belief, the temporomandibular joint is not a weight bearing joint[12,13] and the mandible is not a lever of the third class.[14] It has been demonstrated in cases under observation for many years that such alteration of condyle posture is acceptable to the patient and physiologically sound. The following case report (M. L.) illustrates this point.

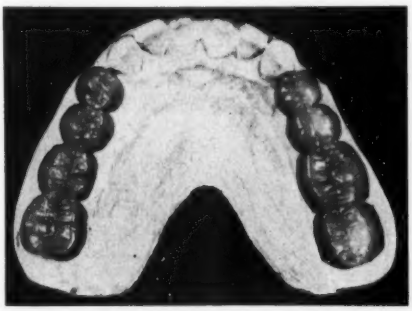

Fig. 4. Case 1. Acrylic full coverage splint units used in treatment of case. (Courtesy Journal of Prosthetic Dentistry)

Extremely important in jaw repositioning is the maintenance of the correct position with no return to the old—in other words, avoidance of a "dual bite." Experience has shown that the corrected position can be maintained and will become entirely involuntary provided mechanotherapy is accompanied by myofunctional therapy.

SUMMARY

The principles and technics of oral orthopedics offer a means of maintaining proper jaw relations and correcting improper relations, and thus are an effective aid in maintaining periodontal health and resolving periodontal disorders.

These principles and technics are important too in orthodontia, prosthodontia and other areas in dentistry; they have a special application in the alleviation of pain, muscle spasm and other disturbances traceable to mandibular imbalance.

It is now seen to be essential, in defining balanced occlusion, to include, in addition to correct tooth-to-tooth relationships, correct jaw relationship and correct muscle balance.

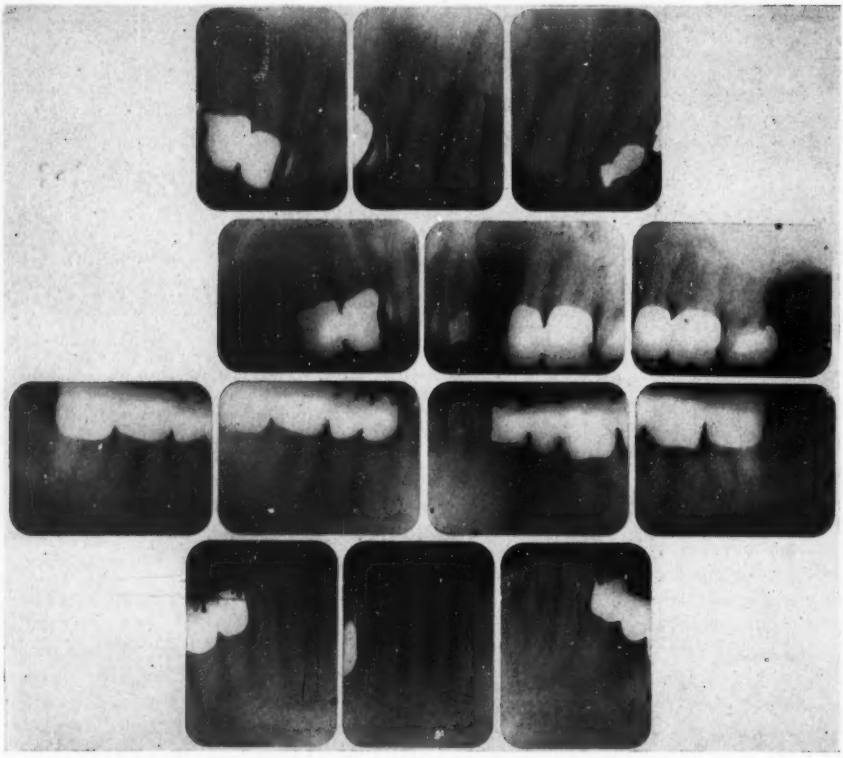

Fig. 5. Case 1. Roentgenograms taken four years after treatment. Note maintenance of level of alveolar bone.

The role of the condyle in mandibular movement is minor and not controlling. The major mechanism in mandibular control is the musculature of the head and neck. The neuromuscular reflexes concerned are under voluntary control and may be conditioned to establish new habit patterns.

Repositioning of the mandible where indicated is accomplished by the use of myofunctional therapy and appropriate occlusal guidance.

Because of its broad anatomical and physiological base and its bearing on general as well as dental health, oral orthopedics has extended the scope of dental medicine. It has also opened the way for further and more effective cooperation with physicians.

CASE REPORT—J. R.

A case presenting an extreme anterior overbite with lingual tilt of the upper incisors and moderate mandibular retrusion is shown in Figures 1 to 5. The patient was a male, 44 years of age, with no significant medical history. Dentally, several teeth were missing; there were gingival congestion and moderate bone loss. The lower right second bicuspid was found to have pulp involvement. It was clear that the excessive overbite posed a serious threat to the dentition. Shortening the anterior teeth by grinding was not indicated since it would not resolve the problem. Apart from the dangers of extensive reduction of tooth anatomy, grinding would do nothing to bring the teeth and jaws into optimal relationship. Conventional "bite opening" also was

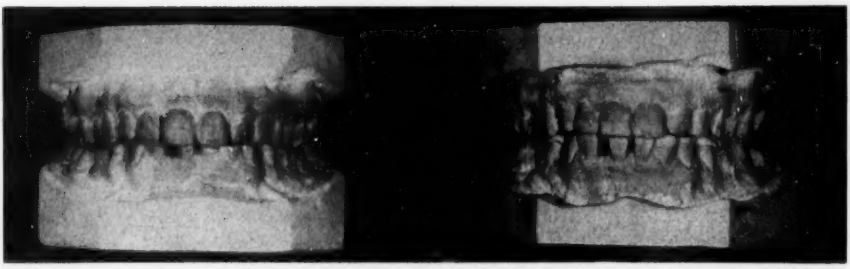

Fig. 6. Case 2. Models showing anterior views of case, (a) before treatment (b) four months later. Note closure of diastema between upper right central and lateral incisors.

not indicated because it too would not bring the teeth and jaws into a correct relationship. In addition it would result in the imposition of additional stress on the posterior teeth and would permit elongation of the anterior teeth, depression of the posteriors or both. To the well known periodontal hazards thus created there would be added a continuation of the original muscle imbalance or creation of a new imbalance.

Correct treatment of the case involved increasing vertical dimension together with forward repositioning of the mandible carried out by appropriate myofunctional therapy. This made possible the reduction of the anterior overbite to an atraumatic level. However, sufficient occlusal contact of the anterior teeth to prevent elongation of those teeth was provided, together with development of the all around occlusal balance needed for maintenance of periodontal health. Quite as important, it made possible the correct alignment of the mandible in relation to the maxilla and the establishment of correct muscle balance.

Figure 1* shows the models of the case when first seen. The initial roentgenograms are shown in Figure 2. The relations developed in treatment are shown in Figure 3. Figure 4 shows the acrylic full coverage splint (crowns joined in a unit on each side) used in attaining the new relation. The bone condition four years later is shown in the roentgenograms in Figure 5.

Note: In selected cases, moderate lengthening of the clinical crowns of posterior teeth produces no periodontal damage providing a state of mechanical and myofunctional balance is established. It will be observed that in this case the condition of the bone around the posterior teeth four years after is excellent.

*Illustrations Nos. 1 to 3 are reproduced by courtesy of Dental Digest, Sept., 1952.

CASE REPORT—M. L.

A case of periodontal disease exhibiting, among other abnormalities, an incipient diastema between the upper right central and lateral incisors, is shown in Figures 6 to 11. The patient, a female, aged 40, reported pain in the temporomandibular joints and clicking on opening and closing the jaws. There was bleeding of the gingivae and slight mobility of the anterior teeth. Medical history was negative. Dental examination revealed moderate anterior over-

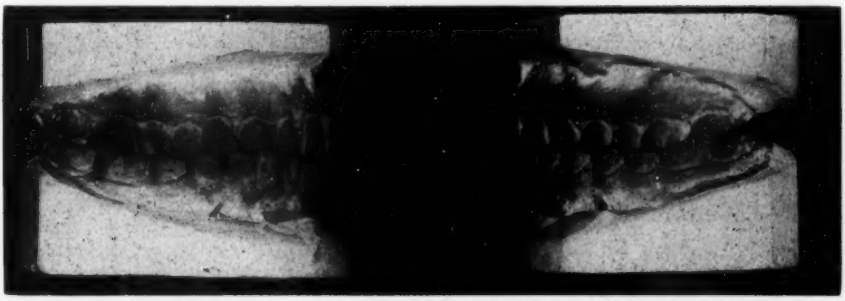

Fig. 7. Case 2. Models showing right and left views of case before treatment. Note overbite and overjet of the anterior teeth.

Fig. 8. Case 2. Models showing right and left views of case after repositioning of the mandible. Note reduction of overbite and overjet. The lower temporary removable splint used in the case is shown.

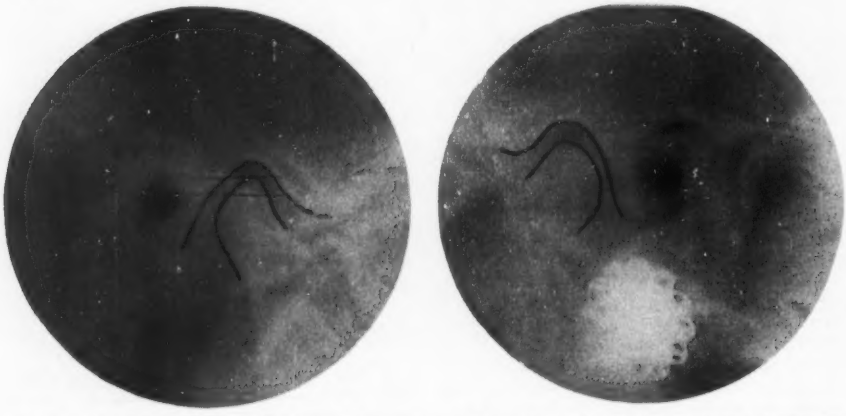

Fig. 9. Case 2. Temporomandibular roentgenograms of case in centric occlusion before treatment. Note slight retrusion of condyles in the fossae. (Note: Figures 9, 10 and 11 have been retouched to facilitate reading of condyle position.)

Fig. 10. Case 2. Temporomandibular roentgenograms of case in centric occlusion after repositioning.

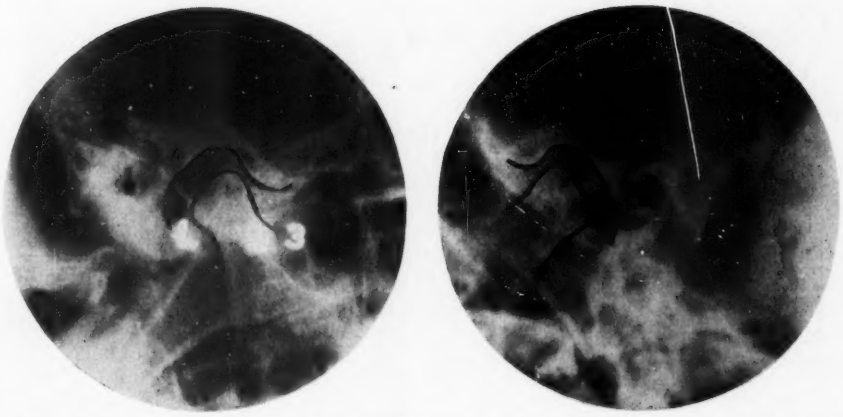

Fig. 11. Case 2. Temporomandibular roentgenograms of case in centric occlusion 18 months after repositioning. Note that the condyle-fossa relations shown in Figure 10 have been maintained.

bite, moderate mandibular retrusion and loss of vertical dimension. The overall picture was one of moderate malalignment but with marked resultant symptoms.

Restoration of lost vertical dimension with reduction of anterior over-bite and forward repositioning of the mandible was indicated. This made possible the necessary reduction of stress on the anterior teeth, correct alignment of the mandible with the maxilla, and the establishment of correct muscle balance.

Figure 6* shows before and after views of the models. Note diastema (left) and its closure (right). The closure occurred spontaneously after relief of the anterior traumatic occlusion. Figure 7 shows right and left views of the case when first seen. Figure 8 shows the relations established by the jaw repositioning (a temporary removable splint with acrylic occlusal surfaces was placed on the lower posterior teeth; a temporary bridge replaced the missing upper right second molar).

After the jaw was repositioned pain in the temporomandibular area and clicking of the joints disappeared. The anterior teeth became firm and the gingivae healthy. Figure 9 shows the condyles in closed position when the case was first seen; there was a slight condyle retrusion. The condyles in closed position after jaw repositioning are shown in Figure 10. The condyle position 18 months later is shown in Figure 11; it was essentially the same as at completion of treatment.

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*Courtesy of Dental Concepts, April-May, 1953.

Case Reports • • • •

Chronic Neurogenic Desquamative Gingivitis with Bullous Formation

by LESTER B. OLDER, D.D.S.,* *Union City, New Jersey*

DESQUAMATIVE gingivitis or desquamative gingivosis as it has more recently been termed, has been investigated thoroughly by several groups clinically and microscopically. In all of these studies a complete description of the pathology involved always includes chronic inflammatory and degenerative changes of the epithelium and connective tissues.[1,2,3,4,5]

It is the belief of these investigators that this condition which is "hypothetically considered a local manifestation of a metabolic disease" has the following causes:[6]

1. Abnormal function of the thyroid gland

2. Interrelations of vitamins and estrogens

3. Disturbance of the endocrine function of the sex glands.

A fourth cause (psychogenic factors) which will be evident from the following case report, should be given emphasis.

It is the intention of this report to present a case of desquamative gingivitis which has been under observation and treatment for a period of two years in which a demonstrable cure has been obtained.

CASE HISTORY

The patient Mrs. O. A. was 34 years of age, 5' ¾" tall, weighing 191 pounds. She had been married for five years and had no children. The patient's dental history revealed frequent visits for prophylactic care and dental checkups.

Fig. 1. Before treatment, uniform band of red inflamed gingivae.

The patient's chief complaint was the formation of blisters on her gingivae wherever any pressure was applied. Fluid which formed in this bulla traveled along the gingival tissues between the epithelium and connective tissues ballooning as it did so. When this elevated epithelium collapsed, the patient was able to remove a large layer of tissue leaving a raw sensitive surface of denuded connective tissue. (Fig. 2.) The patient stated "that she was unable to brush her teeth, or even place food in her mouth without discomfort."

Oral Examination: Oral examination revealed a

Fig. 2. Before treatment atrophied epithelium easily removed, leaving a raw sensitive surface of denuded connective tissue.

*Periodontia Staff, New York University College of Dentistry.

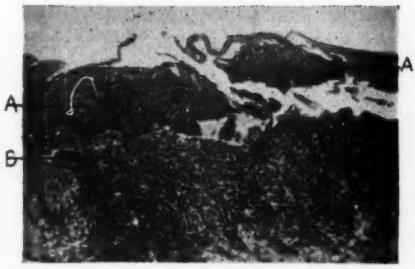

Fig. 3. Photo micrograph, Medium Power, chronic desquamative gingivitis. A—Atrophied stratified squamous epithelium lack of rete pegs, easily detached from underlying connective tissue. B—Subepithelial tissue shows extremely dense collections of small and large mononuclear cell infiltrate.

highly inflamed gingival mucosa which began at the gingival crest affecting the buccal and labial surfaces extending about 4 mm apically from the cervical margins of the teeth. It appeared as a uniform band of red gingival tissue. A line of demarcation was to be noted where the inflammatory gingivae ended and the alveolar mucosa appeared normal. (Fig. 1.) The oral cavity at this time was in a state of uncleanliness with debris, calculus, and materia alba very much in evidence. Little or no periodontal pocket formation was found.

The patient was highly emotional and apprehensive during the examination of the oral cavity.

Roentgenographic Examination: Revealed good alveolar bone support in general with some slight horizontal bone loss in the molar regions.

Physical Examination:[7] Physical examination revealed:

1. The presence of a small anterior cervix, with several small polyps extruding from the os.

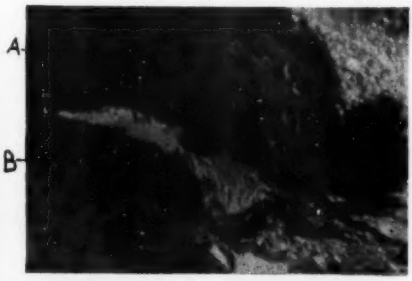

Fig. 4. Photomicrograph, high power, Chronic Desquamative Gingivitis. A—Degenerating atrophied epithelium. B—Subepithelial connective with extremely dense mononuclear cell infiltrate.

2. Extreme overweight.

3. Small papilloma on left side of nose.

4. Sterility problem of 4 years' duration.

The N.Y.U. Dental-Medical Questionnaire[8] was filled out by the patient and its evaluation indicated strong tendencies toward emotional instability.

Laboratory findings:
Urine analysis—Normal
Complete blood count—Normal
Glucose Tolerance—Normal
Urea Nitrogen—13.1 mg%
Cholesterol—268 mg%
Hemogram—Essentially negative with normal differential
17 Ketosteroid—7.6 MGM/24 hours

Impression: On the basis of laboratory and physical findings, the diagnosis is as follows:

1. Obesity, exogenous in origin

Fig. 5. During treatment, consisting of Stillman's method of tooth brushing, with application of modified vitamin A and D ointment. Note tissue tone improvement.

2. Cervical polyps.

3. Possible hypoadrenocorticoidism. This is based upon the surprisingly low 17-ketosteroid and the associated hypoplasia of her genital system. I believe that the patient also can be considered as having hypothyroidism in a mild degree.

Recommendations: 1. 65 mgm thyroid a day in the form of Proloid.

2. A high protein diet almost to the total exclusion of carbohydrates.

3. The cervical polyps should be removed.

4. The patient should be reevaluated within six months to a year concerning the possibility of hypoadrenocorticoidism.

Fig. 6. During treatment, photomicrograph, medium power, utilizing modified vitamin A and D ointment with modified Stillman method of toothbrushing. A—Well differentiated stratified squamous epithelium, lacking to a great degree in hornification. B—Rete pegs and intact basement membrane. C—Subepithelial connective tissue shows no significant changes.

Tentative Diagnosis: A diagnosis of chronic desquamative gingivitis was made. Pemphigus and lichen planus were excluded by differential diagnosis.

Local Treatment, Phase 1: Active treatment consisted of scaling, curettage and occlusal equilibration.[9] An ointment with the following formula was prescribed to be applied three times daily.

Vitamin A water soluble . . 200,000 units
Oil of peppermint 65
Pyrabenzamine Ointment 2% q.s. 60.

The patient was also instructed to take 100,000 units of water soluble Vitamin A daily.

For the next twelve months the patient was treated once a month at which time she was given a thorough scaling and curettage. Calculus which formed readily because of the patient's inability to maintain adequate oral sanitation necessitated the frequent prophylactic care. During this period of treatment several other medications, topically and systemically, were tried for varying periods of time to determine their value as adjuncts. These included pantothenic acid ointment, hydrocortone acetate ointment and cortisone tablets. This therapy produced no improvement.

A biopsy was performed at this time in the area of the upper left cuspid.

Gross Specimen: Specimen consists of a flat grayish white portion of tissue, 1 cm. by ½ cm. in area removed from the oral cavity.

Microscopic Examination: Shows portion of gingivae partially lined by atrophied stratified squamous epithelium which has undergone thinning out with elimination of rete pegs. The epithelium in areas has separated from the underlying connective tissue. Subepithelial tissue shows extremely dense collections of small and large mononuclear cell infiltrate. Deeper, the connective tissue is hyalinized, homogeneous pink staining, and devoid of cells.[10]

Diagnosis: Chronic Desquamative Gingivitis.

Treatment, Phase 2: Local Treatment. At this time a modified dental ointment was prescribed. Continuation of thorough massage to the gingival tissue was deemed advisable in order to produce further stimulation which in turn would result in increased circulation and nutrition to the affected area. The increased circulation would further aid in the regeneration of epithelium.

The modified dental ointment formula follows:[11]

Vitamin A water soluble . . 2,000,000 units
Vitamin D . . . . . 1,000,000 units
Benzocaine 15%
Aquaphor q.s. 60.0

The patient was instructed to apply the ointment four times daily, massaging it vigorously into the gingival tissue before brushing the teeth. Similar applications

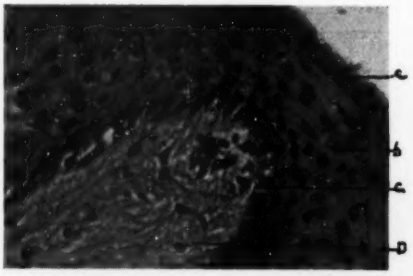

Fig. 7. During treatment, photomicrograph. A—Lack of hornification. B—Deep staining large nuclei of young epithelial cells. C—Intact basement membrane. D—Normal loose subepithelial connective tissue.

were made after toothbrushing and before retiring. This modified ointment provided an anesthetic agent in the form of benzocaine which enabled the patient to increase greatly the amount of stimulation and massage to the gingival tissue. The local application of high concentrations of Vitamin A and D should aid in establishing the integrity of the formation of epithelium. The elements contained in the ointment are incorporated in a water soluble base in order to enhance the local absorption possibilities.

The patient was instructed to begin brushing with a soft toothbrush, using the modified Stillman method which included gingival massage.

Medical Treatment: 1. Surgical removal of cervical polyps

2. Strict reducing diet, high in protein and low in carbohydrates with ½ grain of thyroid three times daily.

3. Multi-vitamin therapy

Three months after this phase of dental and medical treatment was instituted the patient stated that she had observed a marked decrease in the number of blisters that had formed since her last monthly prophylactic visit. The patient lost 17 pounds during this period.

In the following six months, a progressive improvement was noted in tissue tone, color, and physical reaction. The patient's mental outlook has kept pace with these changes. She is now able to brush her teeth and massage her gingival tissue with a hard brush. She can eat and masticate foods painlessly without any signs of bleeding. No bullae have formed for over 8 months.

Fig. 8. After treatment, utilizing Vitamin A and D ointment and increased stimulation with Charters method of toothbrushing, note increased hornification and stippling of gingival tissue.

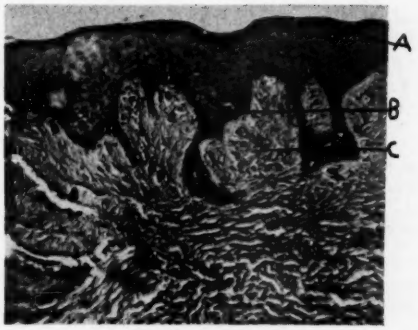

Fig. 9. After treatment, photomicrograph, medium power, utilizing vitamin A and D ointment, and increased stimulation with Charters method of toothbrushing. A—Increased hornification of epithelium. B—Well differentiated stratified squamous epithelium with intact basement membrane. Rete pegs present. C—Normal loose subepithelial connective tissue.

Biopsy procedure was repeated at this time in the region of the upper right lateral-cuspid area. The following is the histopathologic report:

Gross Specimen: Portion of gingival tissue appeared as a flat grayish white surface, measuring 1 cm by ¾ cm in area.

Microscopic Examination: Portion of tissue lined by well differentiated stratified squamous epithelium lacking in hornification. Rete pegs present. Nuclei of epithelial cells large and dark staining, denoting young tissue cells. Subepithelial connective tissue shows no significant changes.

Diagnosis: Normal young gingival tissue with relative lack of hornification.

It would be desirous to have a protective hornified layer of epithelium. Therefore it was decided to change the method of toothbrushing to the "Charters method" to provide increased stimulation and massage and endeavor to produce hornification of the epithelium.

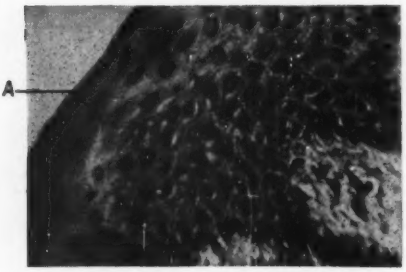

Fig. 10. After treatment, photomicrograph high power. A—Well differentiated hornified stratified squamous epithelium of gingival tissue.

In the following two months, a change in the color and tone of the gingival tissue was evident. The tissue had become pink, non-hemorrhagic, stippled and normal in appearance. Bullae were not present and have not been observed for the past ten months. A biopsy was taken at this time from the region of the upper left lateral-cuspid area and the following is the histopathologic report:

Gross Specimen: Portion of tissue having a grayish-white appearance 1½ cm by 1 cm in area, removed from the gingiva.

Microscopic Findings: Shows well differentiated hornified stratified squamous epithelium. Rete pegs present.

Subepithelial layer of connective tissue shows no significant change.

Diagnosis: Gingival tissue (normal).

The patient is still under periodic prophylactic care. She maintains her vitamin A and D therapy locally and vitamin A therapy systemically, as well as proper oral sanitation. She is still on her diet and has lost 35 pounds to date. She is very happy about the result and is no longer depressed. Her mental attitude has improved tremendously and her cooperation is excellent.

SUMMARY AND CONCLUSIONS

A report of a case of Chronic Neurogenic Desquamative Gingivitis with bullous formation has been presented which required several years of intense therapy to produce a satisfactory result. This case clearly illustrates the need for medico-dental cooperation and understanding. It is apparent that cases classified as desquamative gingivitis have the following possible interrelated causes:

1. Nutritional imbalance
2. Disturbance of endocrine function of sex glands
3. Allergic reactions
4. Psychogenic factors
5. Poor oral sanitation.

This case accentuates the importance of improving or eliminating altogether the psychogenic and systemic factors. The oral condition often contributes to and complicates the problem because of the annoying symptomatology. A local approach if followed intensely may often contribute to the desired result by reducing the effects of the psychic factors. The use of an anesthetic ointment medicated with high concentrations of Vitamin A and D possibly aids in establishing the integrity of the epithelium of the gingivae. The use of this ointment with massage and increased stimulation appeared to give this patient substantial relief. In this case microscopic examination showed the contributing effect of gingival massage in regeneration of the gingival tissue.

I would like to express my sincere appreciation to the following men:

Samuel Charles Miller, D.D.S., Professor and Chairman of the Periodontia Department, New York University College of Dentistry, for his valuable suggestions during the progress of this case.

Gerard Alonzo, M.D., the patient's personal physician, for his excellent cooperation during the treatment of this case.

Theodore Lite, B.S., D.D.S., for his excellent cooperation during the treatment of this case as well as for preparing the microscopic reports and the photomicrographs for publication.

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Case Report—Non Surgical Reduction of Dilantin Overgrowth

by HERBERT BRICKEN, D.D.S., *Silver Spring, Md.*

THE treatment to control overgrowth of the gingiva associated with the use of Dilantin Sodium (diphenylhydantoin sodium) poses a difficult problem in dental practice. The object of the dental treatment is to control the gingival enlargement without stopping the administration of the drug. A further object, if it can possibly be attained, might be to keep the psychic trauma of the further manifestation of his condition from unduly upsetting the patient. Thus, if continual surgical retreatments can possibly be avoided this would have a tremendous psychological advantage to the patient.

The hyperplastic changes seen associated with the use of dilantin may be extremely extensive, sometimes covering the entire crown of the tooth. Surgical measures may be the only possible treatment where the tissue has been enlarged for a long time and is quite extensive. However, if initial treatment with surgery is required every effort could be made from then on to prevent the necessity for surgical retreatments.

An added difficulty is the patient cooperation factor, as in some cases the patient is unwilling or even unable to do the extensive home treatment necessary. Careful, patient, repeated coaching in proper home treatment will sometimes bear fruit in the most difficult of cases.

Before presenting an account of the case which illustrates the above points, perhaps a short clinical and histological description of dilantin overgrowth is in order.[1,2,3]

The basic lesions are found at the margin of the gingiva and consist of painless, firm, pale, pink, stippled enlargements which surround the necks of the teeth. They have tiny lobules and unless they are secondarily complicated by inflammation they show very little tendency to spontaneous bleeding. If they are associated with local irritants the lesions may be red, or even blue-red in cases of chronic irritation and venous stasis. Edema may further complicate the appearance of the hyperplasia.

Histologically, the lesions consist of nonspecific hyperplasia of the epithelium and connective tissue. Rete pegs and acanthosis are seen in the thickened epithelium and the connective tissue shows an increase in fiber bundles, cells, blood vessels and intercellular elements. Inflammatory infiltration is commonly associated with

Fig. 1. Before treatment.

these findings.

Complete reduction of the enlargement without stopping the use of the drug is an extremely difficult result to attain and even more difficult to maintain.

The inflammatory portion of the enlargement will yield early in the treatment, the hyperplastic portion will change slowly and in all probability never is completely reduced but can be kept under control in many cases.

The case presented was treated entirely without any surgical procedures and is being maintained without any surgical procedures.

This report does not represent any recommendation that all cases of this nature be treated by any specific method. It is simply a report of one case which was handled completely by scaling, curettage, polishing and home care and illustrates the type of clinical result that can be obtained by this treatment even under the most adverse conditions.

Fig. 2. Radiographs of case.

HISTORY

The patient, a twenty year old white female, presented herself with the complaint of having the feeling that "marbles were growing in her mouth." Her face showed a heavy, flattened appearance of the nose and lips which indicated an endocrine gland dysfunction. Her mentality while not extremely low appeared sluggish, al-

Fig. 3. After 5 months of treatment.

though at times she seemed quite normal. Her skin, hair and eyes were fairly normal. Her body was well proportioned but she was quite short, five feet tall.

Her oral mucosa was normal, her tongue was enlarged. The gingivae were enlarged causing relative pockets averaging 4-5 mm in depth throughout the mouth on the labial, buccal, and lingual. Her bite was open anteriorly, she had a tongue-thrusting habit and a mouth-breathing habit. The anterior teeth were unworn. The posterior teeth showed prematurities in centric and lateral excursions.

The gingivae showed inflammation at the margins and bled upon palpation. The enlargements were firm generally but did show some signs of edema superimposed

over the hyperplasia. These tissues were not very tender and some stippling was evident. Minute lobulation of the gingivae was evident. The upper teeth were all firm but the lower right bicuspids and all of the lower anterior teeth showed slight mobility.

X-ray examination revealed a generalized thickening of the periodontal membrane. The lamina durae appeared thickened around all teeth except the molars. The bone trabeculae appeared normal (Figure II).

The medical history revealed a background of rheumatic fever with heart complications and the patient had been told she was anemic. She has been taking dilantin sodium for four years. Her attacks are well controlled but about six months before she presented herself for treatment she noticed that her "gums were beginning to get like marbles." She is taking iron and liver for anemia.

Laboratory examinations showed no abnormalities in her blood picture or in her urine. Routine urinalysis, erythrocyte count, hemoglobin estimation and leucocyte count and differential examination were done. Diet analysis showed that she was eating a rather well balanced diet.

Treatment consisted of scaling, curettage, porte polishing, continual coaching in home care. Vaseline was given for nightly use because of the mouth breathing. Home care consisted of an attempted combination of modified Stillman's and Charter's methods with a hard, two row, natural bristle brush with rubber cups and rubber tips used for further stimulation. She was quite cooperative, but her coordination was poor and her brushing, while enthusiastic was not as definite as would be ideal. She spent about forty minutes in her home care routine, about ten minutes four times daily. Some occlusal equilibration was done.

The before picture, Figure I, shows her mouth upon presentation for treatment. After five months of weekly treatments, interrupted from time to time by her failing to appear for several weeks, the result seen in Figure III was recorded. She has maintained this for four months without treatment. At no time has the dilantin been reduced.

The gingival tissue has lost most of the signs of inflammation, edema is reduced, the tissue does not bleed upon palpation. The gingival enlargement is greatly reduced and, while the gingival architecture is far from ideal, it has improved immensely. The relative pockets average 1 mm but the gingival attachment is still high on the crown.

SUMMARY

A case of dilantin overgrowth was presented. This case was treated by scaling, curettage, polishing and home care.

A reduction of the gingival enlargement was obtained which, while far from an ideal result, still was of great clinical interest.

Further treatment, including gingivoplasty would be of use in obtaining an ideal result.

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Union of the Enamel and Gingival Epithelium

by H. A. ZANDER, M.S., D.D.S.,* *Minneapolis, Minnesota*

AN article[1] using the above title presents the findings associated with two artifacts occurring in the preparation of tooth and periodontal tissues for microscopic studies. The conclusion is drawn that there exists "a stronger attachment between enamel and epithelium than between enamel and dentin."

I wish to present a photomicrograph (Fig. 1) from an area of epithelial attachment to the enamel. Depending upon the area in this section from which we might make an enlargement, we could demonstrate either an attachment of the enamel matrix to the dentin at "A," or to the epithelium at "B." Figure 2 is a photomicrograph from another specimen where an artifact "B" split the enamel matrix "C" away from the dentin "A," and at the same time the epithelium "D" is split from the connective tissue "E," showing "how firm the epithelial attachment is." To draw conclusions about the "firmness of the epithelial attachment" from such photomicrographs could be misleading to the practicing periodontist who might not be too familiar with the occurrence of artifacts in the preparation of histological specimens. The type of evidence presented here and by the quoted paper[1] does not

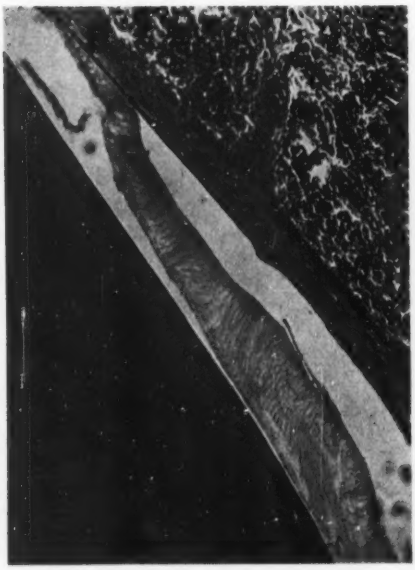

Fig. 1. Photomicrograph of a bucco-lingual section from the buccal crevice of an upper dog molar. At "A" enamel matrix is continuous with dentin matrix. At "B" enamel matrix is continuous with the epithelium. (Mag. X145)

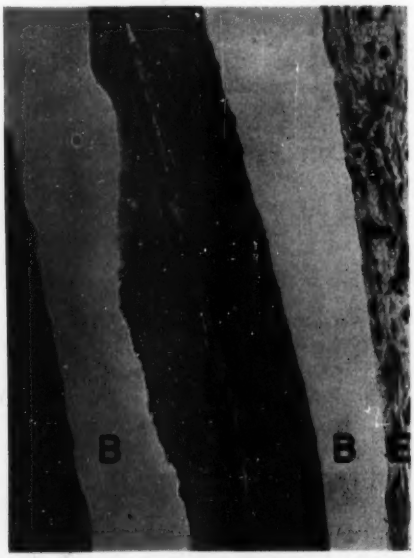

Fig. 2. Photomicrograph from the buccal gingival crevice of an upper dog molar. "A" dentin, "B" artifact, "C" enamel matrix, "D" epithelium, "E" connective tissue. (Mag. X230)

*Professor and Chairman of the Division of Periodontology, School of Dentistry, University of Minnesota, Minneapolis 14, Minnesota.

warrant a clinician's relying on "the firmness of the epithelial attachment" in e.g. probing a clinical pocket. Undue force during such a procedure obviously will *not* detach the enamel from the dentin or the epithelium from the connective tissue before the epithelium gives way from the enamel. Evidence that the attachment of the epithelium to the enamel of the functioning human tooth is not a *strong* or *firm* union will be forthcoming.[2,3,4]

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INTERNATIONAL DENTAL FEDERATION

43rd Annual Meeting 14th-20th, August 1955 in Copenhagen, Denmark

A good attendance from all over the world is expected to the 43rd Annual Meeting of the Fédération Dentaire Internationale to be held in Copenhagen 14th-20th August 1955. The venues of the Meeting will be The Danish Parliament Building in Christiansborg Castle, The Royal Dental College, and The Copenhagen University Institute of Anatomy. Simultaneous translation into English, French and German will be provided throughout business and scientific meetings.

The F.D.I. will hold General Assemblies of Delegates representing many National Member Associations on Monday 15th August and on Saturday 20th August. Sunday 14th August through Friday 19th August there will be meetings of the F.D.I. Council and Commissions as well as an extensive scientific program (Demonstrations, Films, Lectures by international experts, Panel Discussions on Scientific Subjects, Hospital Dental Services, Dental Education, and Armed Forces Dental Services). A special group meeting (workshop) of experts in periodontology is being planned for Sunday 14th August. Subjects at the latter meeting will be "The Epithelial Attachment" and "Biochemistry of Periodontal Tissues."

One whole day will be devoted to a tour of North Sealand including visits to historical places (Elsinore Castle etc.) and there will be a number of receptions by invitation of The City of Copenhagen, the Copenhagen Dental Society, the International Dental Federation and others. There will also be a special program of entertainment for accompanying ladies.

Registration before 1st May 1955 will be at normal fees while registration between 1st May and 15th July will be at increased fees. Hotel reservations will be made by the Organizing Committee if the undersigned Secretary is notified before 1st May 1955. Applications for hotel accommodations received between 1st May and 15 July 1955 can only be complied with to the extent made possible by the total allocation of hotel rooms for the annual meeting. No accommodation can be provided after 15th July 1955.

For full information, enrollment forms and hotel reservation forms please write to W. Riis Klausen, 1, Alhambravej, Copenhagen V. Cable address: Interdentaire Copenhagen. Telephone: Eva 4616.

Editorials • • • •

"TURN ON THE LIGHT . . ."

Readers of this Journal are cognizant of the importance of good oral health to the well being of all individuals, so will be gratified to learn that the American Public Health Association at their last annual meeting (October 1954) passed the following resolution:[1]

> "WHEREAS, the available epidemiological surveys have indicated that some form of periodontal disease affects the community as a whole, and becomes the principal cause of loss of teeth in later life, and
>
> WHEREAS, injurious effects in remote areas of the body may result from periodontal disease more frequently than from other forms of oral disease, and
>
> WHEREAS, the control of periodontal disease represents the next major activity of dental public health, be it therefore
>
> RESOLVED, that the American Public Health Association goes on record as urging dental schools, the National Institute of Dental Health, and other such agencies as may be interested in this health problem to develop and support the immediate promotion of research on methods of prevention and control and on the epidemiology of periodontal disease."

This action is most commendable because it calls the seriousness of periodontal disease to the attention of an important health group, many of whose members are not well acquainted with dental problems.

Thanks are due to Dr. C. O. Dummett and others responsible for drafting and presenting the above resolution to the A.P.H.A. Members of the Academy of Periodontology, and as a matter of fact all dentists, are urged to use their influence to keep reminding all health groups, including dental societies, of the importance of controlling periodontal disease.

"Turn on the light so that the people can find their way."

GROWING PAINS

It is pleasing to note the steady expansion of the circulation of the Journal of Periodontology, but this wider circulation poses many problems. The number of subscriptions, orders for back issues, reprint orders, inquiries, piled on top of the problem of reading and preparing manuscripts for publication, is making the work of the editor less and less enjoyable. We hope, however, that the influence of the Journal will continue to expand, so we won't complain too much.

The majority of the readers of the Journal now are general practitioners of

[1]American Journal of Public Health 44:1610 December 1954.

dentistry with only a small percentage of them dentists who limit their practice to periodontics. Even a smaller percentage of readers are interested in basic research problems on periodontal tissues. For this reason the editor attempts to select a series of articles for each issue, some of which will be of interest and value to each reader. This issue also includes a partial report of the 40th annual meeting of the American Academy of Periodontology which was held in Miami last November. It is recognized that the majority of our readers are not members of the Academy, but it is the editor's belief that this material will be of interest to all readers. We hope that everyone will read of the activities of the Academy, and wish to invite all dentists to attend future Academy meetings as a guest or a member. Furthermore, we urge every dentist who may be so inclined to write the chairmen of the committees listed, sending in ideas and comments for consideration.

Obviously not everything published in all issues will be of interest to all readers, but judging from comments and the expanding subscription list we are batting a fair average.

GOING TO THE SPRING CONFERENCE OF THE AMERICAN ACADEMY OF PERIODONTOLOGY?

One of the more enjoyable, and at the same time, instructive dental meetings held annually is the Spring Conference of the American Academy of Periodontology. This year the meeting is to be held in Ann Arbor, Michigan in the comfortable quarters of the W. K. Kellogg Foundation Institute May 2, 3 and 4. Speakers include:

Thomas H. Weller, M.D., Richard Pearson Strong, Professor of Tropical Public Health and Head of Department, Harvard School of Public Health.

Edward P. Cawley, M.D., Head of Department of Dermatology and Syphilology, University of Virginia.

Muriel C. Meyers, M.D., Associate Professor of Internal Medicine, School of Medicine, University of Michigan.

Robert E. Moyers, D.D.S., Professor and Head of Department of Orthodontics, University of Michigan, School of Dentistry.

Samuel Charles Miller, D.D.S., Professor of Periodontia and Chairman of Periodontia Dept., New York University, College of Dentistry.

Lewis Fox, D.D.S., Assistant Clinical Professor of Dentistry, Columbia University, School of Dentistry.

All members of the American Academy of Periodontology are eligible to attend and each member is entitled to sponsor a guest. The fee for this conference is reasonable; may we suggest you write Dr. W. R. Mann, W. K. Kellogg Foundation Institute, Graduate and Postgraduate Dentistry, Ann Arbor, Mich., for details.

American Academy of Periodontology
1954 Reports

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President's Address

by HARRY LYONS, *Richmond, Va.*

I have the honor of addressing you on this occasion to express a note of appreciation, to make a few observations and to offer several opinions bearing on the future of dentistry and dental education with special reference to periodontology and the Academy.

My appreciation for the privilege of holding high office in the Academy is boundless. To be honored by election to our presidency is a decoration that comes to only a few and I would have you know that I am fully mindful of this. It may come as a surprise to many of you that this office involves little or no effort in the way of labor or responsibility. Those who planned the organizational pattern of the Academy showed great wisdom in placing all policy making in the hands of our Executive Council, all programming, budgeting, etc., in charge of able committees nominated by the Council and elected by our General Assembly, all publications as duties of our Editor appointed by the Council, all finances under control of our Treasurer, and all general administration as the responsibility of our Secretary. No president ever had less to do and fewer opportunities for mistakes of judgment and action. At the same time, no president ever enjoyed an official association with a Secretary, a Treasurer, an Editor, and Committees of greater skill, diligence, and dedication to duty. This is evident in the continued growth and usefulness of the Academy, the quality of its Journal, and every arrangement detail for this meeting, from the moment of your registration until the final tap of the gavel adjourns this meeting and the last cookie is eaten on Saturday afternoon! Without detaining you long enough to commend every worker individually, I am sure that you join me in my expression of gratitude and esteem for all of them. They truly merit, individually and collectively, an accolade of our commendation.

This Academy has had a rich history since its founding in 1914 by a small group of inspired, farsighted pioneers. Our history has been the subject of several presidential addresses in former years, and an interesting paper on the subject was prepared for our 25th anniversary by one of our most highly esteemed members, Doctor A. H. Merritt. Recently, Doctor Edward B. Spalding was commissioned to prepare a detailed history of this Academy and we await his manuscript with restless anticipation. It will surely be noteworthy.

We are, at this annual session, celebrating the 40th anniversary of the Academy's noble birth. The program for the entire session is accordingly dedicated to this historical landmark.

The age of 40 has been described variously as the age of discretion and the age when

Given at the 40th meeting of the American Academy of Periodontology, November 4, 1954.

men roar to reassert their virility, the age of continued joy for the bachelor and of increasing despair for the spinster, the age when life begins and the age when it ends for too many through cardiac and other diseases, the age viewed as old by youngsters and as young by the more aged. A span of 40 years is an eternity when the clock and calendar are watched; it is but a brief moment in the history of man and the world. The record of this Academy since 1914 brightens the pages of dental history as few groups or events may claim. It stands as a beaconlight to guide this and many another ship of state in dentistry.

On this 40th anniversary of the Academy's founding we salute the noble men and women who created our organization and dedicated their energies to its development. This Academy stands as a monument to their glory.

In a brief span of 40 years this Academy has been, in great measure, responsible for the elevation of periodontology from its seedling roots to the full stature of a phase of dentistry co-equal with all the other aspects of dentistry. (You will notice that I have carefully avoided reference to periodontology as a specialty and I shall belabor that point at some length.) After such rapid growth and development, it is important that we pause, examine our course to date, and chart future trends in light of experience and associated factors.

In the early years of its development, those who labored so devotedly and skillfully acted wisely in guiding periodontology in the direction of status as a specialty even though it must have been obvious to most of them that this was really not the ultimate objective to be desired. Then, as now, the aspiration was surely to broaden the scope of dental health care to include effective treatment of disease affecting the supporting dental structures as a dental health program fundamental and basic to all of dentistry and an integral part of it. It was, of course, quite natural that this broad concept of dental health care would be originally sponsored by a limited number of farsighted pioneers who had not only a particular interest but also special skills and knowledge. These individuals were decorated by their appreciative contemporaries with the title "specialist" and were used by many general practitioners for reference and the care of their more difficult periodontal problem cases. Several facts soon became quite obvious. First, and of least importance, enough periodontists, as specialists, to take care of the needs or the demands for periodontal care could not be educated and trained in the foreseeable future. Second, the periodontal problems are so intimately related to other dental problems in both cause and effect that a separation of periodontal considerations from other basic dental considerations cannot be accomplished. The close collaboration required between the general practitioner and those who limit their practices to periodontics attests to this fact. At best, the periodontist serves only the role of an *auxiliary* specialist to the general practitioner. This point need not be labored further for the Academy's membership and guests, except possibly to reiterate that every dental procedure short of two complete artificial dentures has a periodontal bearing either for good or bad; hence, attempts to separate periodontics from general dental care is, at best, a questionable expedient.

With the current widespread trend toward specialization, with the existence already of seven dental specialty boards and applications before the Council on Dental Education for at least two more, and with conversations prevailing in certain circles that some of our dental specialties might well be completely separated from dentistry with separate educational programs of their own, it is high time that the entire field of dental care and dental education be re-evaluated critically, objectively, and without any reference to any one or more special interests but only with the *total* welfare of our profession and the *total* welfare of our populace in mind. The threat of fragmentation of dentistry

appears to be a frightening menace. Fortunately, a survey of dentistry is soon to be undertaken. Promoted by the American Dental Association, financed mainly by the Kellogg Foundation, and directed by the American Council on Education, this survey promises to be one of the most fruitful ventures ever undertaken in the field of health care appraisal. It merits our wholehearted support.

In the meantime, it appears that our Academy might take the lead by re-charting its own emphasis in several directions. First, having promoted periodontology in dental education to the point where it is now taught as a subject entity in all our dental schools, we should now stress the *integration* of instruction in periodontics *into* every other phase of dental education. In dental education, as in dental practice, periodontology should be made an *integral* part of operative dentistry, fixed and removable prosthetic dentistry, pedodontics, orthodontics, oral surgery, and principles of medicine. Basic courses of instruction in periodontology as a health science should, of course, be continued and strengthened, but we must also move now to integrate it into the whole of dental care, of which it should be considered the foundation. The famous philosopher Gestalt taught that education and all other human activities should be appraised not so much by weighing its *component* parts as by an evaluation of its *entirety* or whole. The Gestalt point of view is widely supported in the field of general education, and in the vernacular among some educators reference is made to the "gestalt"—the entirety or the whole of a field or endeavor. It must be admitted that, in the finality of things, this alone counts. It would appear to apply to dental care and dental education with equal force.

Second, I believe we may say, with due modesty, that the Academy has made a most important contribution to health care by promoting interest in periodontology among general dental practitioners. To this end, we have welcomed guests to our meetings and originally sponsored the Section on Periodontia of the annual scientific sessions of the American Dental Association. The ADA Section on Periodontia has offered very limited programs and has had small numbers in attendance in recent years while our Academy and its annual programs continue to attract increasing numbers of general practitioners who have the fine interest in periodontology necessary for well-rounded, general dental service. In the latter connection, one may sense a trend in our programming in favor of practice procedures with a *de*creasing emphasis on research and basic science studies related to periodontology. For these more fundamental aspects of our interest, the Academy has sponsored a seminar (usually held at the University of Michigan) as an *auxiliary* activity quite apart from our annual programs. All this needs to be re-evaluated in terms of the future usefulness and activity of the Academy.

It would appear that the following policy revisions might serve all concerned to better advantage in the years ahead:

1. The Academy should, as an official policy, promote anew the ADA Section on Periodontia by offering to assist in the development of excellent programs for the Section, programs that will attract and benefit general practitioners in large numbers. Our annual Academy meetings have attracted a hundred or more visiting practitioners. The ADA Section on Periodontia should attract a thousand or more of the 8 to 10 thousand who attend the annual ADA meetings. Here is a challenge to be met in fulfillment of our Academy's objective of promoting interest in periodontology among all who practice dentistry. In this connection, it should be noted that all special dental organizations, our Academy included, should add or contribute something to our parent ADA organization and not substitute for, or subtract from it. Special dental groups should not compete with the ADA and thereby weaken our central national organization to which we must look for our professional strength in terms of organizational activity.

2. With the clinical aspects of periodontology promoted as just suggested, the Academy could, and should, then seek to fulfill a more difficult mission—that of sponsoring, promoting, and itself engaging in more fundamental studies and research, thereby becoming the well-spring of further development in the science and practice of periodontics. Our programs under such a plan would be of no immediate interest to general practitioners. Our contribution in their direction would be made through cooperative effort with the ADA through its Section on Periodontia, as previously described. On the other hand, our annual programs could then provide for seminar studies in basic science areas related to periodontology, for the presentation and evaluation of original research, for the sponsorship of newer research projects, and for the development of teaching methods and skills especially useful in undergraduate and postgraduate courses in periodontology. These responsibilities are not being met adequately now and they pose a challenge to this Academy for the future.

3. Further membership privileges might be determined in relation to the proposed program, new members being recruited on the basis of interest and ability to participate in such a program.

The suggested changes in emphasis, activities, and programs for the Academy would direct its future development toward that of an academy in the classical sense of that designation and would avoid the pitfall of the Academy becoming, as some have feared, "just another dental society." The Academy would, under the proposed program, probably not grow in membership numbers; it might even find itself soon a smaller organization numerically. These considerations are of little import when measured against the greater, long term usefulness in terms of what an academy—a body of learned men engaged in study and research—can contribute to the sum total of knowledge in our field. The newer knowledge and its clinical application would quickly find its way into practice channels through the suggested relationship with the ADA Section of Periodontia.

These thoughts which I have shared with you here have been conveyed to our Executive Council for critical evaluation and a committee has been named to study them.

I have cherished the opportunity that has been mine the past year or two to study the Academy at close range, to learn more intimately something of the wonderful contributions made by those who founded and guided the Academy to this point, and to walk in the shadow of the noble men and women who preceded me in this office. The memory of these wonderful experiences will most assuredly hearten and sustain me in the wintry chill of the advancing years. For this and for your warm friendship, I am everlastingly grateful.

Report on President's Address

The committee wishes to compliment President Lyons on the masterful and constructive address which he presented and, on behalf of this organization, to thank him for his several suggestions for elevating the Academy of Periodontology to a position of even greater eminence in the field of health science organizations.

Your committee is in hearty accord with President Lyons' suggestions, and would elaborate on them as follows:

1. In order that periodontology shall eventually assume its proper place in the dental health program, more complete undergraduate instruction in periodontology and more effective interdepartmental correlation is indicated in our dental schools. When this is accomplished, the fragmentation of our profession into an increasing number of sub-specialties will be less likely to occur.

2. Relative to the suggested revisions of policy presented in Dr. Lyons' splendid address, your committee is generally in hearty agreement with our esteemed president, and feels—

    1. Relative to the Periodontia Section of the American Dental Association, that our support of this important group of our parent organization might be better effected if we return to the plan of previous years when the programs were the complete responsibility of the Section officers. These longer, more complete programs attracted gratifyingly large numbers of general practitioners as well as those dentists whose interest lay primarily in the field of periodontology. The relatively brief and restricted programs of the combined sections as presented during current American Dental Association meetings are apparently not attracting any large numbers of dentists to attend.

    2. While the committee agrees that the more practical aspects of periodontia should be stressed on the programs of the A.D.A. Section on Periodontia, there is also a continuing need for the presentation of clinical subjects before the Academy, especially as regards new developments in clinical periodontology.

    3. As regards the basic requirements for membership and its privileges in the American Academy of Periodontology, your committee is fully in accord with the suggestion that President Lyons' thoughts be considered by the proper committees.

Again, your committee compliments President Lyons on his scholarly address and thanks the Academy for the privilege of studying this forward-looking, stimulating and progressive contribution to our Academy.—B. O. A. Thomas, Chairman, Raymond Johnson, Dickson Bell.

Report of the 1954 Constitution and By-Laws Committee

The Committee is well aware that the present constitution and by-laws is a model of efficiency for the operation of the Academy with its present form of government. However, during the past year we have received some rather far-reaching suggestions as to the desirability of changing this form of government.

It is felt that the general assembly of the Academy is too large to serve efficiently as a legislative body.

Broadly, the suggested changes are (1) combine the Constitution and By-laws into one document, to be known as the By-Laws; (2) that the Council be increased to 12 members and shall be the governing body of the Academy, assuming both administrative and legislative duties, except the power to elect officers, and to serve as a final court of appeal.

The Committee feels that the suggested changes merit the consideration of the Council, and if it meets with approval, that monies be appropriated for the procurement of professional advice, so that these suggested changes can be presented to the mid-winter meeting for further consideration.

Richard Chace, Delbert P. Nachazel, James P. Hollers, chairman.

Report of 1954 Committee on Education

At the annual meeting at Cleveland, Ohio, October, 1953, the Committee on Education reported to the Council the summary of statements from 34 dental schools on undergraduate, postgraduate and graduate education in Periodontology. During the past year, 9 additional schools replied to the questionnaire. A summary of the findings from these 43 schools is herewith given.

The total number of lectures, seminar and clinic teaching hours were 4,871. This gave an average of 113 hours for the 43 schools.

The lowest number of teaching hours for one of the schools was 20. The highest was 300 hours. 1,716 hours were utilized for lecture or seminar teaching, which made an average of 40 hours per school. There were 3,155 hours assigned for instruction in the clinic, which gave an average of 73 hours for each school. Thirty-six schools used the lecture method and 40 used the seminar. It is obvious that some schools used both teaching technics.

Fourteen of the 43 schools indicated a need for an increased number of teaching hours. Postgraduate or graduate courses were given by 16 of the 43 schools, and 18 schools were planning to set up such courses.

The Committee on Education has also been interested in determining what the curriculum should be for a two year full-time postgraduate course. A suggested outline for such a course is:

TWO YEAR FULL TIME POSTGRADUATE COURSE IN PERIODONTOLOGY

Leading to a Certificate

FIRST YEAR—FIRST TRI.

	MONDAY	TUESDAY	WEDNESDAY	THURSDAY	FRIDAY
A.M.					
9:00	Review of Literature Seminar	Periodontia Clinic	TMJ Musculature & Occlusion Seminar	Periodontia Clinic	Periodontia Clinic
10:30	Research		TMJ Clinic		
P.M.					
1:30	Periodontia Clinic	Applied Anatomy Lecture	Periodontia Clinic	Psychiatry	Laboratory Periodontal Histopathology
2:30		Research		Research	
4:00			Periodontia Lecture		

FIRST YEAR—SECOND TRI.

	MONDAY	TUESDAY	WEDNESDAY	THURSDAY	FRIDAY
A.M.					
9:00	Review of Literature Seminar	Periodontia Clinic	TMJ Musculature & Occlusion Seminar	Periodontia Clinic	Periodontia Clinic
10:30	Research		TMJ Clinic		
P.M.					
1:30	Biologic Principles of Dentistry Seminar	Applied Anatomy Lecture	Biological Principles of Dentistry Seminar	Internal Medicine	Biologic Principles of Dentistry Seminar
2:30		Dento-Medical Interrelation-ship Lecture		Advanced Radiography Lecture	
4:00			Periodontia Lecture		

FIRST YEAR—THIRD TRI.

	MONDAY	TUESDAY	WEDNESDAY	THURSDAY	FRIDAY
A.M.					
9:00	Review of Literature Seminar	Periodontia Clinic	TMJ Musculature & Occlusion Seminar	Periodontia Clinic	Periodontia Clinic
10:30	Research		TMJ Clinic		
P.M.					
1:30	Periodontia Clinic	Principles of Nutrition Lecture	Periodontia Clinic	Internal Medicine	Laboratory Periodontal Histopathology
2:30		Method of Scientific Research in Dentistry Lecture		Principles of Surgery Lecture	
4:00			Periodontia Lecture		

SECOND YEAR—FIRST TRI.

	MONDAY	TUESDAY	WEDNESDAY	THURSDAY	FRIDAY
A.M.					
9:00	Review of Literature Seminar	Orthodontia Lecture & Clinic	TMJ Musculature & Occlusion Seminar	Periodontia Clinic	Treatment Planning Seminar
10:30	Research		TMJ Clinic		Periodontia Clinic
P.M.					
1:30	Periodontia Clinic	Hospital Rounds	Periodontia Clinic	Dermatology Clinic	Fixed Splinting Clinic
3:30		Research		Clinical Pathology Conference	
4:00			Periodontia Lecture		

SECOND YEAR—SECOND TRI.

	MONDAY	TUESDAY	WEDNESDAY	THURSDAY	FRIDAY
A.M.					
9:00	Review of Literature Seminar	Orthodontia Lecture & Clinic	TMJ Musculature & Occlusion Seminar	Periodontia Clinic	Treatment Planning Seminar
10:30	Research		TMJ Clinic		Periodontia Clinic
P.M.					
1:30	Periodontia Clinic	Hospital Rounds	Periodontia Clinic	Dermatology Clinic	Fixed Splinting Clinic
3:30		Research		Clinical Pathology Conference	
4:00			Periodontia Lecture		

SECOND YEAR—THIRD TRI.

	MONDAY	TUESDAY	WEDNESDAY	THURSDAY	FRIDAY
A.M.					
9:00	Review of Literature Seminar	Orthodontia Lecture & Clinic	TMJ Musculature & Occlusion Seminar	Periodontia Clinic	Treatment Planning Seminar
10:30	Research		TMJ Clinic		Periodontia Clinic
P.M.					
1:30	Periodontia Clinic	Hospital Rounds	Periodontia Clinic	Dermatology Clinic	Fixed Splinting Clinic
3:30		Research		Clinical Pathology Conference	
4:00			Periodontia Lecture		

It is the opinion of the committee that the outline should be sent to all schools of the United States and Canada requesting suggestions for their approval or modification of the contents. This outline could be used as a guide for those schools who are at present developing a full-time postgraduate curriculum in Periodontology.

On the basis of the reported information, future activities of the Committee on Education might consist of setting up the minimum requirements that seem essential to teach Periodontology to undergraduate students. The results of such a study could be presented to the Council on Dental Education of the American Dental Association.

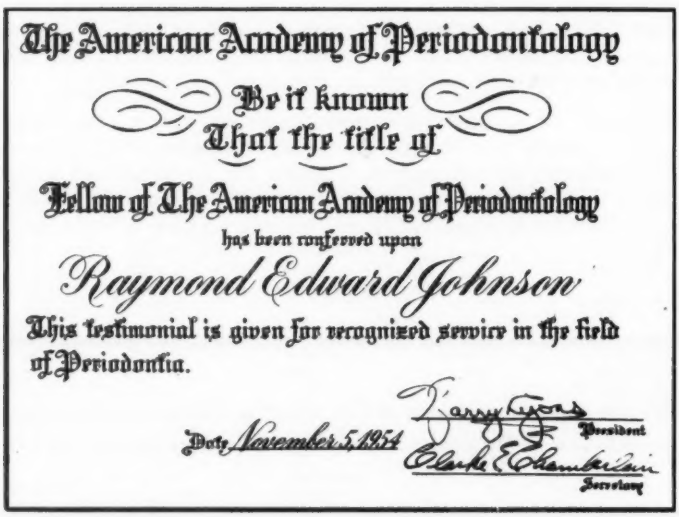
Dorothea F. Radusch, Clarence N. Sanger, Frank E. Beube, Chairman.

We are sorry to report that Dr. Clayton Gracey, well known periodontist of Birmingham, Michigan, died on April 2, 1955.

Dr. Raymond E. Johnson Honored by Academy

The title of Fellow of the American Academy of Periodontology was conferred on Dr. Raymond E. Johnson of St. Paul, Minnesota, in Miami in October 1954. Dr. Johnson was honored for his long and valued services to the Academy and to dentistry.

Dr. Johnson is shown responding to the citation, and his certificate is reproduced below.



The American Academy of Periodontology

Be it known
That the title of

Fellow of The American Academy of Periodontology

has been conferred upon

Raymond Edward Johnson

This testimonial is given for recognized service in the field of Periodontia.

Date *November 5, 1954*

President

Secretary

AMERICAN ACADEMY OF PERIODONTOLOGY

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Robert G. Kesel ..... President-Elect
Clarke E. Chamberlain ..... Secretary
James E. Aiguier ..... Treasurer

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James E. Aiguier ..... Treasurer
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Donald A. Kerr 1955
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Harry Wood 1956
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(Replaced by Harry Lyons, December, 1954)
Harold J. Leonard, Sec'y.-Treas. 1955
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B. O. A. Thomas 1956
Raymond E. Johnson 1957
Clarke E. Chamberlain, Chairman 1957

AMERICAN ACADEMY OF PERIODONTOLOGY

Committees for 1955

STANDING COMMITTEES OF GENERAL ASSEMBLY

Constitution and By-Laws

James P. Hollers, Chairman ..... 1 year 1955
D. P. Nachazel ..... 2 years 1956
Richard Chace ..... 3 years 1957

Membership

Hunter S. Allen, Chairman ..... 1 year 1955
Dickson G. Bell ..... 1 year 1955
Isador Hirschfeld ..... 2 years 1956
Edward B. Spalding ..... 3 years 1957
Carlos H. Schott ..... 3 years 1957

Nomenclature and Classification

Joseph L. Bernier, Chairman ..... 1 year 1955
Hermann Becks ..... 2 years 1956
Henry Goldman ..... 3 years 1957

Education

Frank E. Beube, Chairman ..... 1 year 1955
Clarence N. Sanger ..... 2 years 1956
Dorothea F. Radusch ..... 3 years 1957

Research in Periodontology

Irving Glickman, Chairman ..... 3 years 1957
S. Leonard Rosenthal ..... 2 years 1956
Sigurd Ramfjord ..... 1 year 1955
(in place of Harold Ray)

Scientific Sessions

B. O. A. Thomas, Chairman ..... 2 years 1956
Donald A. Kerr ..... 3 years 1957
Evert Archer ..... 1 year 1955
(in place of Robert Kesel)

Ethics

Mildred Dickerson, Chairman ..... 1 year 1955
Craft A. Hopper ..... 2 years 1956
Lowell N. Peterson ..... 3 years 1957

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Committee for Spring Conference of the A.A.P.

Elizabeth M. Downie, Chairman
Joseph Barkley Harry Wood

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(Arrangements for Annual Meeting)

Local Arrangements

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Robert W. Rule, Jr.
J. E. Ziegler
E. G. Baker
L. E. Breese
Harold Santos
Craft A. Hopper

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D. P. Nachazel
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Beryl T. Ritchey

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Registration

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R. E. Stoker
M. J. Calbeck
R. Roseberry

Properties

Harold Santos, Chairman
Earl Gilmore
Jack Curran
A. A. Ariaudo

Round Table Luncheon

Co-Chairmen

Justin D. Towner Gordon Agnew

Clinic

Sigurd Ramfjord, Chairman
H. E. Grupe
J. W. Neilson
F. G. Everett

Entertainment

J. E. Ziegler, Chairman
H. V. Jones
Robert Hutson
J. I. Ingle

Ladies Activities

Mrs. Harold Ray, Chairman
Mrs. Robert G. Kesel
Mrs. Dickson G. Bell
Mrs. B. O. A. Thomas
Mrs. Lowell Peterson
Mrs. Robert W. Rule, Jr.
Mrs. Robert Hutson
Mrs. Earl Gilmore
Mrs. E. G. Baker
Mrs. L. E. Breese

Announcements • • • •

THE AMERICAN INSTITUTE OF DENTAL MEDICINE

The next Annual Meeting of the Institute will take place at the Desert Inn, Palm Springs, California, October 23 to 27, 1955. The faculty will consist of:

Maury Massler, D.D.S., M.S., University of Illinois, Chicago: Effects of filling materials on the pulp; Sterilization of dentin; Tissue changes during ageing.

Valy Menkin, M.D., Temple University Medical School, Philadelphia, Pennsylvania: Inflammation, bacterial invasiveness and immunity; Chemical factors liberated by injured cells in inflammatory exudates; The anti-inflammatory problem with particular emphasis on the mechanism concerned, as induced by corticosteroids.

Hans Selye, M.D., University of Montreal, Montreal, Canada: Recent progress in the study of stress as applied to dental medicine.

Reidar F. Sognnaes, D.M.D., Ph.D., Harvard School of Dental Medicine, Boston, Massachusetts: Relative significance of cellular and chemical remodeling of bones and teeth as revealed by radioactive isotopes; The complexity of the caries problem suggested by recent observations in experimental animals; The clinical implications of experimental caries research.

Wendell L. Wylie, D.D.S., University of California, San Francisco, California: Tooth guidance and factors influencing the rate of eruption.

All seminar lecturers will participate in a round table forum discussing the application of their subject to the practice of Dental Medicine. Applications and full information may be secured from the Executive Secretary, Miss Marion G. Lewis, 2240 Channing Way, Berkeley 4, California.

AMERICAN BOARD OF ORAL PATHOLOGY

The new officers of the American Board of Oral Pathology elected at the Annual Meeting in Chicago are: Colonel Joseph L. Bernier, President; Dr. Hamilton B. G. Robinson, Vice-President; and Dr. Donald A. Kerr, Secretary-Treasurer.

The next examination for certification by the American Board of Oral Pathology will be held at the Armed Forces Institute of Pathology, Washington, D. C. the week-end of December 11, 1955. Inquiries relative to the examination and the Board should be directed to Dr. Donald A. Kerr, University of Michigan, School of Dentistry, Ann Arbor, Michigan. Completed applications should be in the office of the Secretary before October 15, 1955 to qualify for the December examination.

COLUMBIA UNIVERSITY
SCHOOL OF DENTAL AND ORAL SURGERY

A course on the Principles of Occlusion, PD 429, consisting of lectures, seminar conference and group participation, and clinical demonstrations will be given by Professor Lewis Fox and associates on May 11, 12, and 13, 1955, at Columbia University School of Dental and Oral Surgery.

This course is designed to present the fundamentals of occlusion of the teeth in relation to the physiology and pathology of the periodontal supporting tissues. The entire course will be directed toward correlating the principles of occlusion to clinical criteria and therapeutic endeavors.

The fee is $150. Application must be made no later than April 20, 1955. For further information write to Postgraduate Division, School of Dental and Oral Surgery, Columbia University, 630 West 168th Street, New York 32, N. Y.

UNIVERSITY OF PENNSYLVANIA
PERIODONTOLOGY—GRADUATE COURSE

The Graduate School of Medicine of the University of Pennsylvania and the Riesman Dental Clinic, Beth Israel Hospital, Boston, Massachusetts, jointly announce their affiliation in sponsoring a graduate two year training program in Periodontology. The program will be under the Chairmanship of Dr. Henry M. Goldman.

The course consists of one academic year at the Graduate School of Medicine with emphasis in the sciences basic to dentistry and clinical periodontology. The second year at Beth Israel Hospital will be devoted primarily to the clinical practice of this specialty and to research. A certificate will be awarded at the end of the second year. This plan meets the standards of graduate education of the American Board of Periodontology. It is designed for those interested in teaching as well as specialized practice. Upon completion of the curriculum and a thesis, students would be considered by the Graduate School of Medicine for the degree of Master of Science.

Enrollment will be limited to six. Applications should be sent to the Office of the Dean, Graduate School of Medicine, University of Pennsylvania, Philadelphia 4, Pennsylvania.

TUFTS COLLEGE DENTAL SCHOOL

The Division of Graduate and Postgraduate Studies of Tufts College Dental School announces a comprehensive two year specialist training program in Periodontology leading to a Masters degree. The course is under the direction of Dr. Irving Glickman and consists of the following three main sub-divisions,

1. Instruction in basic sciences, oral medicine and clinical medicine.

2. Clinical training in Periodontology. Half of the student's time is devoted to the treatment of patients in our Postgraduate Clinic. Aspects of Occluso-rehabilitation, Orthodontics and Prosthetics which pertain to periodontal problems are taught by lecture and seminar.

3. Research. The facilities and guidance of the Oral Pathology Department and other departments of the Dental and Medical Schools, as well as departments in the New England Medical Center Hospital are available for laboratory and clinical research.

For further information address Director of the Division of Graduate and Postgraduate Studies, 136 Harrison Avenue, Boston 11, Mass.

UNIVERSITY OF MINNESOTA SCHOOL OF DENTISTRY

The University of Minnesota School of Dentistry and Center for Continuation Study announces its Tenth Annual Seminar for the Study and Practice of Oral Medicine. Sessions will be held at the Continuation Center in Minneapolis on September 23 and 24, 1955.

Subjects to be discussed will be of special interest to the general practitioner as well as those interested in specialized practice. A complete program of participants will be outlined at a later date.

Further details and information may be obtained by writing to the Center for Continuation Study, University of Minnesota, Minneapolis 14, Minnesota.

FALL PERIODONTOLOGY COURSES AT COLUMBIA UNIVERSITY

Four postgraduate courses in Periodontology are offered at Columbia University, School of Dental and Oral Surgery of the Faculty of Medicine.

A full-time program covering a period of two academic years and a half-time program covering a period of four academic years lead to a Certificate of Training in Periodontology. Correlation of the fundamental sciences to clinical practice is emphasized.

Of the two part-time courses, one course will be given one full day a week from September 21, 1955 to May 2, 1956; the second course will be given one half day each week from September 21, 1955 to May 2, 1956.

For further information write to Postgraduate Division, School of Dental and Oral Surgery, 630 West 168th Street, New York 32, N. Y.

ARPA MEETS IN VENICE

The Bureau of the Arpa International has requested an organized film program for the coming 14th International Arpa congress in Venice, September 1955. Members or friends of the academy are invited to show scientific films at the congress. Application should be made before the 15th of July, 1955.

Please write for details to:

Dr. H. R. Mühlemann, Professor of Operative Dentistry and Periodontology
University of Zurich School of Dentistry, Zurich 28, Switzerland

TWO COURSES IN PERIODONTICS ANNOUNCED FOR 1955

by Dr. Balint Orban

Theory and Practice of Periodontics—June 21-25

This course will cover the basic sciences in lecture periods and clinical demonstrations will include prophylaxis, home-care techniques, gingivectomy and post-operative care, flap operation, occlusal analysis and adjustment.

Occlusion—July 2-6

This course will consist of lectures and demonstrations with each participant doing an occlusal diagnosis and adjustment.

For further information, please write to: Dr. Balint Orban, 629 N. Nevada Avenue, Colorado Springs, Colo.

THE EMORY UNIVERSITY SCHOOL OF DENTISTRY

presents a one week postgraduate participating course in

PERIODONTOLOGY

Dates: May 23rd to 27th, 1955. Class Limit: 10. Tuition: $100.00. Registration: Monday, May 23, 1955; 9:00 A.M. Dental School Building, 106 Forrest Avenue, N.E., Atlanta 3, Georgia.

Half of the time of this course is devoted to clinical practice by the participant. The remainder of the curriculum will consist of lectures and seminars.

The lectures and seminars will present the histopathology of periodontal disease, the rationale for diagnosis and treatment, the determination of prognosis, the technics employed in periodontal therapy, occlusal equilibration and diagnosis of diseases associated with the oral cavity.

In the clinical practice, the participants will do gingivectomy, gingivoplasty, occlusal equilibration, subgingival curettage and oral physiotherapy.

Mail application and tuition fee to Emory University School of Dentistry, 106 Forrest Avenue, N.E., Atlanta 3, Georgia.

POSTGRADUATE TRAINING IN PERIODONTIA AND ORAL MEDICINE

NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

The following postgraduate courses in Periodontia and Oral Medicine will be offered at the New York University College of Dentistry and will be given by Dr. Samuel Charles Miller and Staff:

To acquire clinical skills associated with periodontal therapy as well as an awareness of some problems of periodontal pathology.

1. Three weeks, Full time, Daily.
   Monday through Friday, 9:30 A.M. to 4 P.M.
   June 6, 1955 to June 24, 1955.
   Tuition including books and instruments: $382.50.
   Class limited to 24.

For preparation towards specialization and teaching.

2. Two years, Full time, Daily.
   Monday through Friday, 9:00 A.M. to 4 P.M.
   September, 1955 to May, 1957.
   Tuition including books and instruments: $2,150.50.
   Class limited to 6.

3. Four years, Half time, 2½ days weekly.
   September, 1955 to May, 1959.
   Tuition including books and instruments: $2,150.50.
   Class limited to 6.

For comprehensive preparation in periodontia.

4. One year, Full time, Daily.
   Monday through Friday, 9:00 A.M. to 4 P.M.
   September, 1955 to May, 1956.
   Tuition including books and instruments: $1,145.50.
   Class limited to 6.

5. Two years, Half time, 2½ days weekly.
   September, 1955 to May, 1957.
   Tuition including books and instruments: $1,145.50.
   Class limited to 8.

An introduction to the study of some of the more recent advances in the field of periodontia.

6. Ten sessions, Intermittent, Evening and afternoon.
   October, 1955 to December, 1955.
   First four lecture sessions—Wednesday, 7:00 P.M. to 9:00 P.M.
   Last six clinical demonstration sessions—Wednesdays, 4:00 P.M. to 6 P.M.
   Tuition including books and instruments: $202.50.

Further information concerning the courses listed above can be obtained by writing to the Secretary, Postgraduate Division, New York University College of Dentistry, 209 East 23rd Street, New York 19, New York.

THE UNIVERSITY OF ALABAMA SCHOOL OF DENTISTRY

A three-day refresher course in DIAGNOSIS AND TREATMENT OF DEFORMITIES OF OCCLUSION: With Special Emphasis on Selective Grinding to be given June 3, 4, and 5, 1955, under the direction of Louis A. Cohn, D.D.S., F.A.C.D., Associate Clinical Professor of Dentistry, School of Dental and Oral Surgery, Faculty of Medicine, Columbia University, New York, New York; and Associate Professor of Post Graduate Prosthetics, Tufts Dental School, Boston, Massachusetts.

This course is especially designed to acquaint the general practitioner with the basic principles involved in occluso-rehabilitation. The subject matter will explore the classification of types of occlusion, the problem of centric, the functional and nonfunctional movements of the mandible as they influence diagnosis and treatment planning, and various methods of treatment and a technique of selective grinding. Lectures and seminars will be illustrated with slides, casts and black-board talks. Clinical evaluation of several problems of occlusion will be made and the selective grinding treatment of one case demonstrated.

The tuition fee for the course will be $100.00, and the class enrollment will be limited. For further information, write to

Joseph P. Lazansky, M.D., D.M.D.
Associate Dean
Director of Postgraduate Education
University of Alabama School of Dentistry
1919 Seventh Avenue South
Birmingham 3, Alabama

COLLEGE OF DENTISTRY, OHIO STATE UNIVERSITY

The College of Dentistry, Ohio State University, has arranged a series of postgraduate courses to be presented during the first six months of 1955 for the general practitioner of dentistry and those interested specialists. Most of these courses are given for five consecutive days—Monday through Friday—and the enrollment of each course is limited in order to permit intimate teaching. The fees vary from $35 to $100 per course.

On May 9 to 13 a new course is being presented, "Periodontology and Diseases of the Mouth on the Basis of Oral Pathology," by Dr. J. R. Wilson and Dr. H. B. G. Robinson. Further information and application may be secured from the College of Dentistry, Ohio State University, Columbus 10, Ohio.

TUFTS COLLEGE DENTAL SCHOOL

DPG 704—Occluso-Rehabilitation I—Lecture and Seminar Course

Monday through Friday

Sept. 19-23, 1955 inclusive, 9 a.m. to 5 p.m.

Tuition $250.00

Dr. Louis Alexander Cohn

This will be a didactic presentation consisting of illustrated lectures devoted to the principals governing the theory and practice of Occluso-Rehabilitation. Diagnosis and treatment planning will be stressed. Seminars in case management will be conducted and implemented by models, radiographs, and motion pictures.

DPG 705—Occluso-Rehabilitation II—Special Participation Course

Every other week on Friday and Saturday 9 a.m. to 5 p.m.

Seven Months beginning October 7, 1955

Tuition $1,000.00—Enrollment limited to twelve

Dr. Louis Alexander Cohn

This is a participation course in which each student will start and complete a patient who requires all phases of Occluso-Rehabilitation. Dr. Cohn will personally supervise all the clinical and laboratory activities. The inter-relationship of Occluso-Rehabilitation and Periodontal Therapy will be stressed. It is recommended that each participant bring a patient from his private practice. However, clinic patients will be available. Occluso-Rehabilitation I is a prerequisite for this course.

BEG YOUR PARDON . . .

We are sorry to report that the word "not" was left out of the second sentence of the first paragraph on page 19 of Dr. Glickman's article on "Trauma and Pocket Formation" which appeared in the January issue of THE JOURNAL OF PERIODONTOLOGY. We apologize to Dr. Glickman and the readers for this omission; one must admit that this three-letter word does change the meaning of the paragraph!

The sentence should read, "The fact that periodontal pockets did not occur when trauma from occlusion was introduced under such circumstances emphasizes the negative role of trauma in the initiation of periodontal pockets."

Also, the name of Dr. Alvin C. Hileman of Seattle, Washington was inadvertently left off the Roster of Diplomates published in the January issue.

Letter to the Editor • • • •

An editorial entitled "Some Advice on Giving Advice on Nutrition" appeared in the December 1954 issue of J.A.D.A. I find that my views differ with some of the statements made in that editorial as well as in the January 1955 Journal of Periodontology. My version of the problem is contained in the following reply.

It may not be proper to do as the ancient hunter who "ate the heart of his adversary in the belief that it would give him strength and courage." It would be more nutritious, however, to eat organ meats such as kidney, spleen, liver and heart—certainly not of human origin, but of animal origin. This type of food is considered superior to some of our televised and radiotized versions of wheat, whether it be steamed, puffed, crunched or shot out of cannons and then preserved with chemicals that even mice would avoid eating.

The diatribes contained in the editorial should not be slanted towards the few faddists in our profession, but towards the manufacturers of food products. Many food processors have the temerity to influence our children to buy a particular brand of vitiated wheat product that will make them as strong and as virile as cowboys who probably would not even partake of the product. And what about sugar industry advertisements which tell us to eat sugar to reduce?

The editorial criticizes the commercial food faddist for exaggerated claims, but it also asserts that many vendors of vitamins are particularly obnoxious in this respect. I feel that such a comparison is totally unjustified.

Although overeating and obesity are currently the number one dietary and nutrition problem in this country, I know of no instance in which a food company is encouraging a reduction in food consumption. In fact, these companies continue to urge the buying and consumption of more and more food. Their one concession to the recognition of the problem of obesity is their claim, when possible, that their particular product is low in calories. The inference that it can be consumed in unlimited quantities without adding weight is present nevertheless.

The most outrageous practice, however, is the one perpetrated by a group of food companies on children. Devices well known in the advertising field are particularly flagrant on the television. Staring straight at the young child, with the product held high in the air, is the announcer who delivers his commercial with urgency, pressure and "the fear of God." He literally orders the child to run straight to the store to buy the product, or more subtly, he urges the child to exert pressure on his unsuspecting mother. How many children can resist, when they are influenced into thinking that they will not be socially acceptable or will not grow up to be big and strong unless they consume the product? An alternative "gimmick" is the usually worthless trinket advertised for a small charge plus a box top of a certain product. In such cases, the pressure on the parent through the child often reaches an almost uncontrollable pitch, as the sponsor well knows.

Even so called non-ethical drug houses marketing vitamins have rarely stooped to this low level of huckstering. It is a most interesting fact that ever since the appearance of vitamins, criticism has been aimed at their manufacturers. Yet, I know of no one

vitamin manufacturer who has violated good taste as much as some of the companies in the food industry. Through the years there has been cultivated the notion that the food industry is pure and the drug industry is infamous. To us the facts indicate that the reverse may well be true.

It is almost impossible to satisfactorily answer the writer of an editorial by means of a letter. Even though it may be true that "there are many blanks in nutritional knowledge," it may be no less true that available knowledge concerning the cause of periodontal disease and decay contain many more blanks. Yet, we treat these clinical entities every day and perform excellent services for our patients. Our attitude should encompass not only correction of disease entities, but also prevention. It behooves us to know much more about nutrition because it is in this very field that the solution to the causes of periodontal disease and decay may be found.

In a recent issue of the Journal of dental education, J. C. Muhler* states that "Certainly nutrition is applied biochemistry but it appears that it would be difficult to adequately teach students as much nutrition as they must have to practice proper preventive dentistry, and also satisfactorily prepare them in the practiced principles of nutrition, considerable amounts of periodontal disease and dental caries can be prevented. This fact alone seems to justify the inclusion of nutrition as a separate course in the dental Curriculum."

The December editorial of the J.A.D.A. states that "When suggestive oral lesions are present it is desirable for the dentist to seek cooperation of the physician since the possible malnutrition may also involve conditions requiring medical treatment." No one disagrees with cooperating with the physician. However, if the patient has no apparent complicating medical problem, then it becomes increasingly difficult for the dentist who is conversant with oral manifestations of nutritional deficiencies, whether of the tongue, mucosa or gingivae, to refer his patient to a physician for evaluation and treatment of nutritional inadequacies. The dentist is in a dilemma whether to refer the patient to a stomatologist or to a pathologist or to a dermatologist. Nutrition is not the exclusive property of any one specialty, but seems for the present, to be inadequately handled by all the specialties.

It is not necessary in this enlightened day and age to apologize for the theories and disciplines of the evaluation and treatment of nutritional inadequacies. Animal as well as human experimental and clinical evidences for treating the pathologic patterns of nutritional deficiencies are conclusive enough. It is up to the dentist to keep himself informed of the latest aspects of nutrition.

There may be dentists who abuse the use of nutritional supplements just as there may be dentists who prescribe antibiotics indiscriminately. Our patients, however, would derive greater benefits if we dentists, rather than some ill-informed laymen, would prescribe dietary advice.

Harry Roth, B.S., D.D.S., F.A.C.D.
156 East 52 Street
New York 22, N. Y.

*Muhler, J. C., Survey of Status of Biochemistry in the Dental School Curricula, Jour. Dent. Education, 18:4 Nov. 1954.